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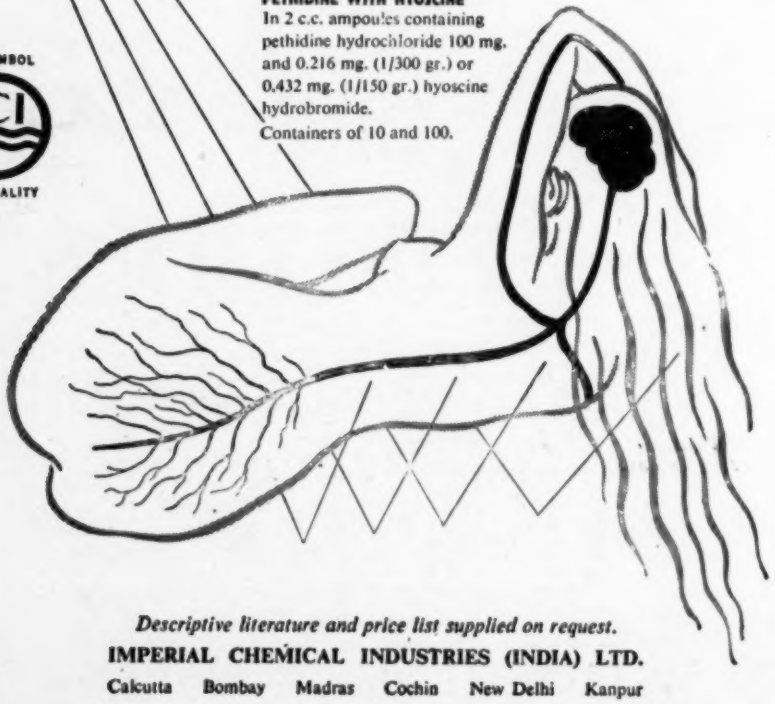

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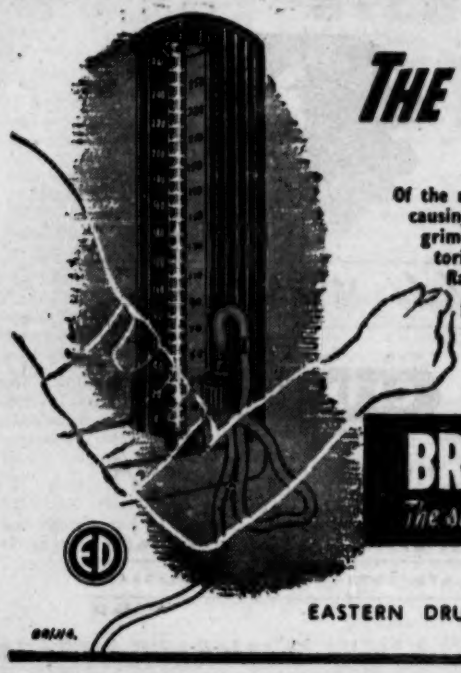

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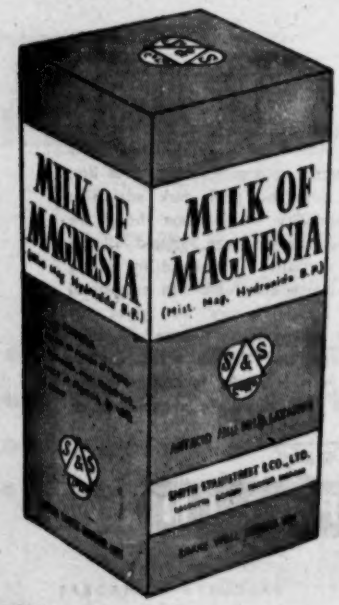

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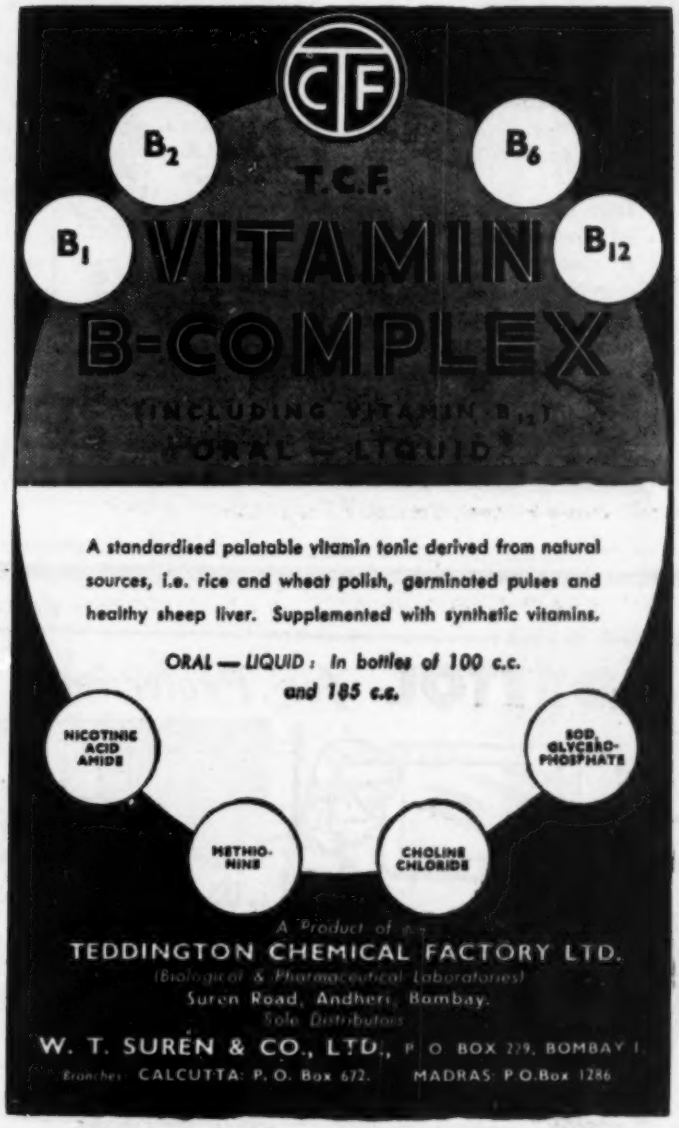
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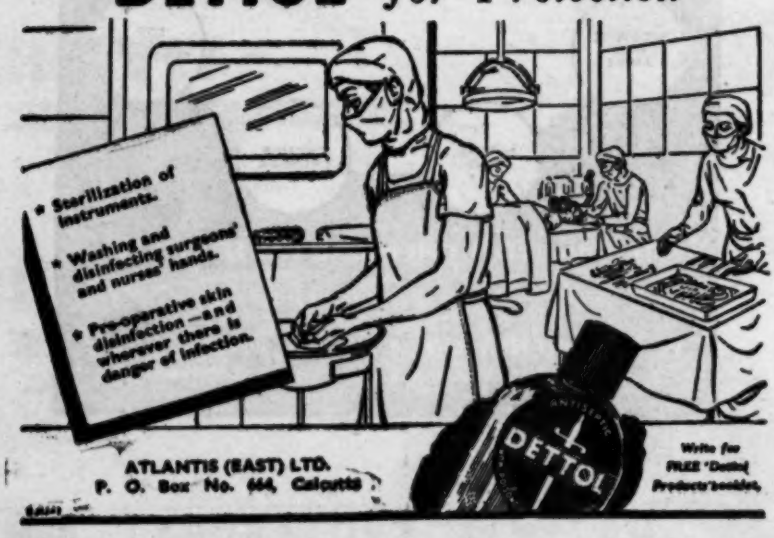
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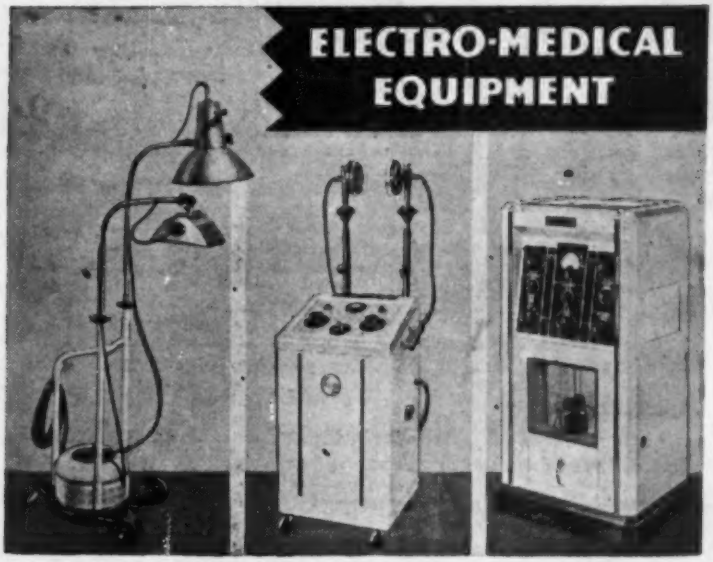

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[1] Britton C. J. C. (1950): Practitioner, 164, 458

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ORIGINAL ARTICLES

BODY FLUID DISTRIBUTION AND ANTIDIURETIC HORMONE IN URINE IN DIFFERENT TYPES OF OEDEMA

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The distribution of the body fluid and the role played by endocrines in the pathogenesis of renal and other types of œdema have been a subject of controversy. Brown and Rowntree (1928) have shown that in acute and in subacute nephritis the plasma volume remains within normal limits except when severe anæmia is present. Litchwitz (1929) observed high plasma volume in early acute glomerulonephritis whereas Nonnebrauch (1921) is of the opinion that in nephritis there may be either decrease or increase in plasma volume depending upon electrolyte concentration, colloid concentration and other factors operating at a particular moment. Waterfield (1931) reported actual fall in plasma volume in nephritic œdema during the formation of œdema and a rise during its subsidence. McClure and his associates (1933) also confirmed the above view. Gibson and Evans (1937) working on nephrotic œdema concluded that in most cases there occurs an increase of plasma volume. Robinson and Farr (1940) demonstrated the presence of an antidiuretic substance in the urine of patients suffering from the nephrotic syndrome.

Rallie *et al* (1944) demonstrated the presence of an antidiuretic principle in the urine of patients suffering from advanced cirrhosis of the liver with oliguria. Perrara (1946) reported an increase of plasma volume in Laennec's cirrhosis. Lichtman (1948) remarked that there occurs a disproportionate increase of the tissue fluid volume over the plasma volume in cirrhosis of the liver.

Mollison (1946) investigated the plasma volume of 6 male and 9 female prisoners in Belsen concentration camp and found an increase of plasma volume of about 50 per cent above normal in nutritional œdema. Henschel *et al* (1947) working on experimental nutritional œdema on human subjects concluded that there was an increase of plasma volume as well as of thiocyanate space in all cases investigated by them. Gopalan (1950) reported the presence of antidiuretic hormone in the urine of his patients suffering from nutritional œdema associated with marked oliguria.

The present investigations were carried out on patients suffering from either renal, cirrhotic or nutritional œdema. Plasma volume and extracellular fluid volume were determined in these patients and an assay of antidiuretic principle in the urine was made in selected cases. Finally, an attempt has been made to correlate the findings in body fluid distribution with the presence of antidiuretic hormone in the urine.

MATERIALS AND METHODS

Investigations were carried out on 30 patients admitted in the Medical Wards of the Nilratan Sarkar Medical College Hospitals during the period of April 1950 to August 1951. Ten of these patients had cirrhotic liver, ten had nephritis and the rest suffered from nutritional œdema. Only those patients in whom the diagnosis was established beyond doubt with the aid of clinical and laboratory findings were studied for the purpose of this investigation. Urinary antidiuretic hormone was estimated on 4 patients each of cirrhosis and malnutrition and on 10 patients with nephritis.

PLASMA AND TISSUE FLUID VOLUME DETERMINATION: *Method*—Plasma volume was determined by the " dye method " of Gregersen using 0·7 per cent solution of T-1824 in pyrogen-free distilled water. One ml. of coloured plasma obtained 10 mins. after injection of 5 ml. of the dye solution was mixed with 5 ml. of saline. The colour was compared with a standard prepared from 0·9 ml. of control plasma mixed with 0·1 ml. of 1 in 50 dilution of the dye and 5 ml. of saline.

Extracellular fluid volume was determined by the method of Gregersen and Stewert (1939). Sixteen ml. of 5 per cent solution of sodium thiocyanate was injected into the arm vein (through the same needle used for injecting T 1824). The concentration of thiocyanate was determined in a sample of serum

collected from the opposite arm vein one hour after the injection. To 5 ml. of trichloracetic acid filtrate was added 1 ml. of ferric nitrate and the colour was compared with a standard prepared from the same ampoule.

A photo-electric colorimeter was employed for both the plasma volume as well as for the thiocyanate estimation.

Interpretation—In order to assess the result of our findings we estimated the plasma volume and interstitial fluid volume in ten normal adults. The results are expressed in Table 1.

TABLE 1—SHOWING PLASMA VOLUME AND TISSUE FLUID OF TEN NORMAL INDIVIDUALS

Case No.	*Initials*	*Age Sex*	*Body weight in Kg.*	*Total plasma volume in litres*	*Plasma volume per Kg. of body weight in ml.*	*Total tissue fluid volume in litres*	*Tissue fluid per Kg. of body weight in ml.*	*Tissue fluid: plasma volume*	*Other hæmatological data*	
									P.C.V.	*R.B.C. in million*
1	K	30F	39	1·9	48·7	6	154	3·16	38	4·5
2	J	40M	43	2·3	53·5	7·7	180	3·4	45	5
3	FJ	38M	49	2·9	59·2	10	204	3·5	44	4·9
4	TD	28F	45	2·3	51·1	7·7	171	3·3	39	4·4
5	MS	45M	52·8	2·7	51·1	8·4	159	3·1	44	5
6	R	45M	60	2·7	45	8·9	148	3·3	45	5·1
7	SM	40F	51	2·6	50·9	7·9	155	3·04	42	4·8
8	SB	35M	40	2·2	55	6·4	160	2·9	45	4·8
9	SD	20M	36	1·95	54·2	7·5	208	3·8	42	4·9
10	BD	30F	40	2	50	7·7	197·5	3·9	40	4·5
			...	Mean	52·2		174·3	3·34		

These findings indicate that in healthy individuals plasma volume ranges between 45 ml. and 59·2 ml. per Kg., with a mean of 52·2 c.c. per Kg. of body weight. This figure is in close agreement with most other published figures on the subject.

It may be pointed out that the main difference in blood volume of the two sexes is accounted for by the greater corpuscular volume in the males. We have therefore omitted the corrections necessary for the variation of the sex of the individual patients.

The tissue fluid volume was found to vary between 148 ml. and 208 ml. per Kg. of body weight with a mean of 174·3 ml. per Kg. The volume of tissue fluid in an individual is equal to 3·34 times the plasma volume. These standards have been employed in the evaluation of the data presented here.

ANTIDIURETIC HORMONE: *Method*—The method advocated by Rallie *et al* (1944) was followed.

Twentyfour hours urine, collected under toluene and acidified with acetic acid, was evaporated to about 80 ml. at room temperature below electric fans. It was then dialysed in cellophane bags for about 8 hours and the volume was made upto 100 ml.

Groups of four albino rats weighing between 120 and 200 gms. served as test animals. The rats were hydrated to 5 per cent of their body weight with tap water introduced into the stomach with a rubber catheter. One ml. per 100 gms. of body weight of processed urine was then injected intraperitoneally to these rats and the antidiuretic potency of the urine was calculated from the inhibition of water-diuresis.

Two groups of 4 rats were used for each experiment and the mean of the two experiments was taken to be the result.

Interpretation—The first four experiments were done on groups of hydrated rats, receiving 1 ml. of water per 100 gms. of body weight intraperitoneally instead of processed urine. The excretion time of 25 per cent and 50 per cent of ingested water was noted as shown in Table 2.

TABLE 2

		Time (min.) for 25% excretion	*Time (min.) for 50% excretion*
Expt. 1	...	(1) 60	90
		(11) 60	75
Expt. 2	...	(1) 60	75
		(11) 75	120
Expt. 3	...	(1) 75	90
		(11) 75	105
Expt. 4	...	(1) 60	75
		(11) 60	90

It will be seen that hydrated rats excreted 25 per cent of water ingested within 60 to 75 mins. and 50 per cent of the water ingested within 75 to 120 mins., then no injection of normal or abnormal urine was given.

RESULTS

Result of assay of normal urine for antidiuretic hormone: Twentyfour hours urine from four normal subjects were collected and "processed" in the way described above. Groups of 4 hydrated rats were then given intraperitoneal injections of 1 ml. of processed urine per 100 gms. of body weight and the time required for excretion of 25 per cent and 50 per cent of ingested water was then noted. The results are shown in Table 3:

TABLE 3

Case No.	Sex	Urine volume 24 hrs. in ml.	Sp. gr.	Excretion time (min.) 25%	50%
*1	M.	1450	1014	75	150
				90	165
2	F.	1600	1010	75	135
				90	165
3	M.	1100	1016	90	170
				105	185
4	M.	1200	1012	75	165
Average	...	...	...	85	165
Range	...	...	...	75 to 105	135 to 185

This shows that normal urine causes slight antidiuresis. Average time of 25 per cent excretion was 85 min. and 50 per cent excretion was 165 min. as compared with 60 to 75 min. and 75 to 120 min. respectively when no urine was given.

In the present investigation 18 samples of urine collected from patients suffering from nephritis, cirrhosis and nutritional œdema were examined for antidiuretic activity.

TABLE 4—SHOWING INCREASE OR DECREASE OF PLASMA VOLUME AND INCREASE OF TISSUE FLUID AND ANTIDIURETIC HORMONE EXCRETION IN URINE OF CASES OF NUTRITIONAL OEDEMA, CIRRHOSIS OF LIVER AND NEPHRITIS

Serial No.	Initials	Age Sex	Body Weight in Kg.	Total plasma volume in litres	Per cent increase or decrease of plasma volume	Total tissue fluid in litres	Per cent increase of tissue fluid	Tissue fluid: plasma volume	Antidiuretic hormone in urine Excretion time (min.) 25%	50%
					NUTRITIONAL OEDEMA					
1	BK	25M	35·5	2·4	+32	9·06	45	3·7	...	...
2	BB	51M	40	3·1	+49	13·2	89	4·3	...	...
3	KD	55M	44	2·9	+26	11	43	3·8	60	90
4	SD	35F	35	2·5	+38	11·2	83	4·5	...	...
5	Sati	38M	45	2·6	+13	9·6	23	3·7	75	105
6	MLD	35M	43	2·5	+13	10·4	37	4·2	...	...
7	PD	32F	36	2·1	+11	10·1	61	4·8	...	...
8	S	30F	30	1·9	+19	8·3	58	4·4	60	90
9	SP	30M	38	2·8	+40	8.3	27	2·9	...	...
10	MM	35M	43	2·4	+ 9	10	33	4·2	90	105
					CIRRHOSIS OF LIVER					
1	SCK	32M	53·5	3·6	+33·3	14·1	51	3·9	...	...
2	RPS	31M	46	2·6	+ 8·3	11	37	4·2	...	...
3	LND	40M	40	3·4	+60	13·5	93	3·9	...	...
4	AD	50M	42	3	+37	13	78	4·3	...	...
5	GM	40M	54	3·8	+36	15	59	3·9	90	120
6	PS	30M	46	3	+25	13·6	70	4·5	...	...
7	SBB	40M	53	3·4	+30	15·5	67	4·6	150	255
8	RM	30M	50	2·7	...	12·3	41	4·5	135	180
9	AB	50M	42	2·6	+18	10·8	48	4·1	...	...
10	MD	40M	42	...	...	..	...	...	105	165
					NEPHRITIS					
1	CC	22F	42	2	− 9	11·2	54	5·6	270	300
2	CD	20M	40	2·1	...	11	57	5·2	165	300
3	S	20F	45	2·1	− 8	12·2	56	5·8	165	275
4	K	30F	45	3	+30	14·9	91	4·9	105	255
5	B	35M	58	2·6	−13	14·3	41	5·5	270	330
6	AUR	25M	67	2·4	−31	13·8	19	5·7	155	255
7	SC	60M	...	...	...	...	...	...	150	330
8	AKS	25M	...	...	...	...	...	...	105	255
9	A	33M	...	...	...	...	...	...	270	360
10	MB	18M	...	...	...	...	...	...	90	135

The results fell into 3 categories as follows:

25 per cent excretion below 85 min. and
50 „ „ „ „ 165 min.

Normal antidiuretic activity
No excess of antidiuretic hormone.

Those cases in which the excretion times were prolonged beyond these limits had high antidiuretic potency in the urine. Cases of the latter group were divided into "moderate" and "severe" class after the method advocated by Rallie *et al.*

Moderate antidiuretic activity:
25 per cent excretion between 85 and 155 min.
50 „ „ „ „ 165 and 215 „

Marked antidiuretic activity:
25 per cent excretion above 155 min.
50 „ „ „ „ 215 „

These results for the body fluid distribution and urinary antidiuretic hormone of each group of patients are presented in Table 4.

1. NUTRITIONAL OEDEMA: Plasma volume and tissue fluid volumes were estimated in ten patients of this group. The average increase in plasma volume for this series was found to be 25 per cent above normal. The lowest level for the tissue fluid volume was 10 per cent above normal, and highest was 40 per cent above it, the average increase being 24·8 per cent.

The ratio tissue fluid/plasma volume was determined in each case. It was found to vary between 2·9 and 4·8 and the average value for this ratio was 4. This ratio in the normal individuals investigated here varies between 2·9 and 3·8 with an average of 3·34. It is obvious from our findings that in our series almost all the patients suffering from nutritional œdema had increased plasma volume and tissue fluid but the increase was proportionate in so far as the ratio tissue fluid/plasma volume did not alter significantly from normal.

Antidiuretic hormone assay of urine was made on four patients of this group. The rats excreted 25 per cent and 50 per cent of ingested water in 71·5 min. and 97·5 min. respectively and the excretion was so rapid that the results could be compared to group 1 control experiments in which hydrated rats were given intraperitioneal injection of water only instead of urine. The antidiuretic activity of the urine was thus "subnormal" in these patients. These patients had no "oliguria" at the time of experiment, the 24 hours urine volume ranging between 1,300 and 1,800 ml. a day.

2. CIRRHOSIS OF LIVER: We had nine patients in the group in whom the plasma volume tissue fluid volume estimations were done. The plasma volume was within normal limits in one case and was increased in the others. The lowest elevation of plasma volume was 8·3 per cent and the highest 40 per cent above normal, the average increase being 27·3 above normal. The increase in tissue fluid varied between 13 per cent and 48 per cent above normal, the average increase being 28 per cent.

The average tissue fluid plasma volume ratio in cirrhosis was 4·3, a slight but definite increase above the normal. This indicates that in cirrhosis there occurs a rather asymmetrical increase of fluid in the two body compartments. These findings point to relatively higher increase of tissue fluid than that of plasma volume in cirrhosis.

Urinary antidiuretic hormone concentration was also estimated in four cirrhotic patients the body fluid figures for 3 of whom are available. Moderate antidiuretic activity could be detected in the urine of 3 of the 4 patients investigated. The average excretion time of 25 per cent and 50 per cent of ingested water by the rats in 3 cases in which antidiuretic hormone was positive being 130 min. and 200 min. respectively.

In most of these patients urinary volume was below 1,000 ml. a day. It is interesting to note that in one of these patients the urine was analysed on three successive occasions (Case No. 7 S.B.B.) on the 12th, 22nd and the 3rd of the next month. The urinary antidiuretic hormone was normal on the first two occasions but was high on the 3rd when the urinary volume also fell to about 450 ml. from 1,200 ml. day. The patient subsequently became anuric and died after a few days.

3. NEPHRITIS: The body fluid estimations were done only in 6 patients with nephritis. In most of these cases, the plasma volume tends to sink below normal or remains within the normal limits. The tissue fluid, on the other hand, shows a remarkable increase in nephritis, the average increase of tissue fluid for these six patients being 3·6 above normal. This fall in plasma volume associated with elevation of tissue fluid volume altered the ratio tissue fluid/plasma volume significantly, which was now 5·3 as compared with 4·3 in cirrhosis and 4 in malnutrition, and 3·34 in normal individuals.

This indicates that in nephritis there occurs selective accumulation of fluid in tissue spaces and the capacity of the blood to hold water is diminished.

The figure for the urinary antidiuretic hormone in patients with nephritis seems to be very interesting. Assay of antidiuretic hormone was undertaken in the urine of ten nephritis patients and excepting one (Case No. 10) who was recovering, high concentration of antidiuretic hormone could be detected in the urine of each of them. The time taken for excretion of 25 per cent and 50 per cent of ingested water by the hydrated rats was much prolonged as compared with the other groups. The average of nine estimations in which a positive result was obtained being 184 min. and 297 min. for the excretion of 25 per cent and 50 per cent of water respectively.

Presence of high concentration of antidiuretic hormone in the urine of the same patient in more than one sample could be detected. Thus in Case No. 1 (C.C.) the estimations were done twice, in Case No. 6 estimations were done thrice. Excess of antidiuretic hormone was found in each separate sample. In most of our nephritis patients urinary volume could not be related to the antidiuretic hormone concentration in the urine.

The relationship between the tissue fluid/plasma volume ratio and the antidiuretic hormone concentration in urine is expressed in Table 5. It will be seen that high concentration of antidiuretic hormone is found in these cases where the former ratio in increased or, in other words, where there occurs selective retention of fluid in the tissue spaces as in the nephritis patients of our series.

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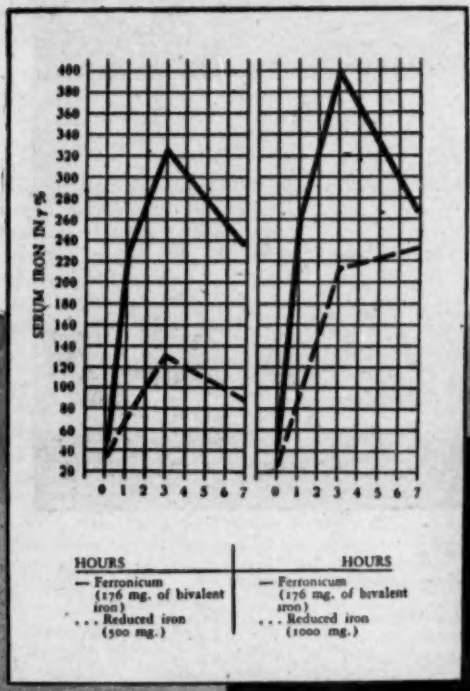

The graph shows the better absorption of Ferronicum as compared to reduced iron. Small oral doses of Ferronicum increase the serum iron level far more than large doses of reduced iron (average from 5 patients). (Jasinsky, Schw. Med. Wschr., 80, 59, 1950)

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TABLE 5

		Tissue fluid: plasma volume	*Excretion time (min.)* 25%	50%	*Antidiuretic hormone conc.*
Normal	...	3·34	85	160	Normal
NEPHRITIS					
CC	...	5·6	270	300	+ + +
CD	...	5·2	165	300	+ + +
S	...	5·8	165	270	+ + +
K	...	4·9	105	255	+ + +
B	...	5·5	270	330	+ + +
Aur	...	5·7	155	255	+ + +
NUTRITIONAL OEDEMA					
KD	...	3·8	60	90	Subnormal
Sati	...	3·7	75	105	,,
S	...	4·4	60	90	,,
MM	...	4·2	90	105	,,
CIRRHOSIS OF LIVER					
SBB	...	4·6	150	255	+ +
RM	...	4·6	135	180	+
GM	...	4	90	120	Normal

DISCUSSION

The unbalanced increase of fluid in the interstitial spaces in nephritic œdema associated with urinary excretion of antidiuretic hormone can be correlated when it is recalled that the activity of the " osmoreceptors " which regulate antidiuretic hormone secretion is controlled by the electrolyte concentration of the tissue fluid which bathes them. Theobald (1937), Verney (1947), and others have shown that antidiuretic hormone secretion is stimulated in all conditions where the salt concentration of body fluid is increased and there is need for water conservation in the body. Such a need occurs in conditions of dehydration or in cases of salt retention. That salt retention play an important role in the pathogenesis of nephritic œdema is known for a long time. Loeb (1933) who studied the problem critically concluded that in nephritis " salts are at first retained in the tissue spaces where they exert osmotic pressure primarily." The same conception of salt deviation from the intravascular to the extravascular compartment was embodied in Fishberg's hypothesis (1944) of " Pre-renal deviation of electrolytes." The work of Gilman and Goodman (1937) throws further light on the relationship between antidiuretic hormone secretion and " salt " present in the body. They administered hypertonic saline by mouth to normal rats and demonstrated that during the diuresis that followed, antidiuretic hormone was excreted in large amounts. When water was administered diuresis occurred but no antidiuretic hormone was secreted. Again, hypertonic saline given to hypophysectomised rats produced diuresis which was characterised by absence of antidiuretic hormone in urine. Walker (1939) confirmed the above findings on diuretic rabbit preparations by administering hypertonic saline by subcutaneous route.

The above discussion establishes clearly the cause and effect relationship between the salt retention in nephritis and the antidiuretic hormone secretion though it must be admitted that the exact mechanism of antidiuretic hormone secretion and its persistence over a long time in nephritis remains obscure at the present moment.

The antidiuretic hormone secreted in nephritis is, of course, compensatory in nature and helps to maintain isotonicity of extracellular fluid.

It has been pointed out by various workers that the œdema of malnutrition cannot always be explained quantitatively by hypoproteinæmia and avitaminosis. Henschel and his colleagues (1947) in a series of well controlled experiments on human volunteers found that œdema appears when the plasma proteins fall by only 0·73 gm. per cent, the albumin globulin ratio remaining constant. They also showed that the plasma volume and tissue fluid volume were both elevated in a more or less proportionate ratio, in relation to the fasting weight but were normal in relation to the normal weight of the individual before fasting commenced. Evidences have been put forward by Henschel, Taylor and their colleagues to substantiate the view that the famine œdema is a reflection of reduction of cellular mass without large change in the absolute amount of body water. They conclude that " famine " œdema is due to relatively excessive hydration of the body."

The patients suffering from nutritional œdema, therefore, can be compared to the hydrated rats of the laboratory which exhibit water diuresis. In other words—the relatively large amount of water in the body of the patients lowers the electrolyte concentration and thereby inhibits the secretion of the antidiuretic principle in the posterior pituitary. In our opinion this explains the rather surprisingly low amount of antidiuretic hormone in the urine of our cases of nutritional œdema. Gopalan (1950), however, found an excess of antidiuretic hormone in the urine of patients with nutritional œdema.

It is probable that the hepatic damage present in many of these cases (Gopalan and Patwardhan 1950) was responsible for impaired inactivation of the hormone and consequent increase of antidiuretic hormone in the urine.

It has been recognised that one of the contributory causes of œdema and ascites in advanced hepatic cirrhosis is high concentration of antidiuretic hormone in the body. Thus Rallie *et al* demonstrated that urinary antidiuretic hormone concentration in cirrhosis of liver varied directly as the severity of œdema and ascites. As mentioned above it has been postulated as being due to failure of inactivation of the antidiuretic

hormone by the damaged liver. Our findings indicate that antidiuretic hormone may be found in the urine of cirrhotic patients who have got excessive œdema and ascites. This observation is in general agreement with the views expressed by different observers.

Summary and Conclusion

Thirty cases of renal, hepatic and nutritional œdema have been studied. The plasma volume, tissue fluid volume and the urinary antidiuretic hormone were determined in these cases. Ten apparently healthy patients from surgical wards were also studied to serve as control.

In renal œdema, the plasma volume tends to be unaltered while the thiocyanate space is remarkably expanded, so that the ratio tissue fluid/plasma volume is considerably higher than normal. This is associated with marked increase of antidiuretic hormone secretion.

In cases of œdema associated with cirrhosis of the liver the plasma volume and thiocyanate spaces were both increased and so the ratio plasma volume/tissue fluid was only slightly higher than normal, urinary antidiuretic hormone excretion was moderately increased in 3 out of 4 cases.

On the other hand, in nutritional œdema, both the thiocyanate space and the plasma volume were considerably increased but the ratio was not significantly changed. There was evidence of a rather subnormal amount of antidiuretic hormone in urine.

It is concluded that secretion of the hormone (either increase or diminution) is determined by the concentration of electrolytes in the tissue fluid and this seems to have an apparent correlation with the tissue fluid and plasma volume ratio.

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INFANTILE CIRRHOSIS OF LIVER

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This disease is unfortunately not uncommon in Madhya Bharat and the surrounding parts of Madhya Pradesh, Rajasthan and the Bombay Presidency. The actual incidence of the disease is difficult to estimate. In our Children Ward which used to comprise of sixteen beds only, two hundred patients were admitted during the period of 12 years from 1938 to 1949.

The ætiology and the pathogenesis of the disease are not definitely known. In spite of the frequency of the disease, opportunities for studying its pathology have been very limited, owing to the extreme difficulty of having postmortem examination on these cases. The present paper is based on the study of the records of the two hundred cases admitted in the King Edward Hospital during the above-mentioned period.

Review of Literature

This disease was first described by Sen (1887) in a paper on "The enlargement of the Liver in children," read in the meeting of the Calcutta Medical Society on the 12th October, 1887. Some of the members present in the meeting pointed out the familial incidence of the disease and Ghose quoted by Sen (1887) thought that

dietetic errors and unhygienic surroundings brought about " a kind of chronic hypertrophy."

Gibbons (1887) first described the morbid anatomy and the pathological histology of the disease. He described it as hypertrophic biliary cirrhosis and thought it was probably due to absorption of some chemical irritant of gastric origin or derived from infant's food setting up inflammatory changes in the bile ducts during the process of their elimination in the bile. It is interesting to note that on a later discussion on the subject, Sen (1890) pointed out the family incidence of the disease and remarked " It is no doubt very difficult to assign any cause which may satisfactorily account for the disease but I may be pardoned for venturing to throw out a hint that some constitutional predisposition or dyscrasia is inherited by the children which under any of the least exciting causes gives rise to the disease."

Pearse (1909) who was then the health officer of Calcutta collected statistics for the year 1907, and gave a clinical account of the disease and concluded that " Taking all things into consideration we can most reasonably conclude that it is a parasitic disease, but whether microbic in origin or due to larger forms, there is no evidence to show."

Mukherji quoted by Rao (1935) pointed out that the disease was more common in " grossly overfed and pampered children in Bengal." On account of the overfeeding the liver cells were overstimulated and as a result, compensatory hypertrophy and enlargement of the liver took place. After some time the liver got exhausted and derangement of the liver cells took place with consequent contraction of the liver. Incidentally he also stated that the disease was not present in any place at a level higher than one thousand feet above sea level. (Indore and the neighbouring parts of the Malwa plateau is about 1,800 feet above sea level).

Mallik (1922) thought that the disease was due to " unwholesome food and faulty digestion, coupled with a predisposition inherited from the mother who was usually found to be in bad health."

Green-Armytage (1926) thought that owing to dietetic deficiency and ill health of the mother the baby obtained a supply of milk from its mother lacking in stimulatory hormones, calcium, iron, phosphorus, iodine etc., such defects culminating in intestinal and hepatic derangement of the infant. He also pointed out that " indiscriminate feeding and overfeeding and lack of normal quantities of essential elements in the milk of animals in India are factors in the incidence of the disease."

Pandalai (1934) considered the disease to be due to faulty feeding in children with congenitally insufficient liver with inadequate defensive powers.

Radhakrishnarao (1935) published a detailed account of the morbid anatomy and histopathology of five cases of infantile liver and concluded that the disease was a subacute toxic condition. He thought that the disease was not what Gibbons thought as hypertrophic biliary cirrhosis and pointed out that the cholangitis which was found in one of his cases was due to superimposed infection. He was of opinion that " a congenital susceptibility of the liver probably prepares the ground for the action of the toxin which produces vascular lesions around the venous tree, necrosis of hepatic parenchyma and replacement fibrosis."

Lahiri (1936) following Opie's technique, on the basis of animal experiments and laboratory investigations on clinical cases, thought that the disease was due to " sub-infection especially by *B. coli* or streptococci, superimposed on already devitalised and overtaxed liver."

Radhakrishnarao (1937) stated that "Investigation into the dietary of children affected with the disease shows no remarkable difference between their food habits and those of normal children of the same age, caste and social position." He thought the importance of dietetic errors in the causation of the disease was generally overstressed.

Prabhu (1940) stated that the family incidence of the disease was between twenty and thirty per cent, and suggested that the Mendelian law of inheritance might be a factor operating in these cases. He opined that infection, intoxication and metabolic strain were the exciting factors. He thought that the use of zinc oxide and insulin were useful in the treatment of these cases.

Kini (1940) drew attention to the congenital factor in the causation of the disease and suggested that " toxin and organisms or both may be carried by blood through the placenta from the mother causing liver damage. He suggested that sulphonamides should be given a fair trial in this disease along with vaccines.

Sarma (1946) suggested that allergy was a factor in the ætiology in some cases of the disease. He reported good results from the use of adrenal cortex. " Adrenal cortex given orally or by injection has given very useful results in cases with allergic basis." Torantil was also used with " considerable temporary improvement " in two of his cases.

Balasundaram (1947) treated one hundred and two cases of infantile cirrhosis of liver with choline dihydrogen citrate and stated that seventy cases recovered. He used 3 to 4 tablets a day. " Sixty two of them were early cases with only slight enlargement of liver, palpable spleen, slight anæmia, phosphate in urine, irritable temper and a slight diminution of weight. All these cases recovered within two weeks. Thirteen cases came in after ascites had set in. Four of these recovered. Three cases came in an advanced state of the disease and all of them ended fatally inspite of treatment."

Krishnarao (1950) is of opinion that "there are two factors which bring about the disease—(*a*) cow's milk, (*b*) *B. coli* infection. Cow's milk lacking in certain

essential food factors especially choline renders liver vulnerable to infection. *B. coli*, which is always present in the intestine, becomes pathogenic and produces a low grade inflammation of the liver resulting in the formation of cirrhosis." He reported encouraging results from stopping cow's milk and feeding children on proprietary milk feeds, choline, vitamin B complex and *B. coli* vaccine. Subsequently he used streptomycin instead of *B. coli* vaccine with good result (choline 15 to 20 grains three times a day).

I have come across very few publications concerning infantile cirrhosis of liver in European and American literature. Hurst thought that "it was extremely doubtful whether such a clinical entity (biliary cirrhosis) existed." Congenital cirrhosis has been described by Rolleston and McNee. These cases were associated with congenital obliteration of the bile ducts but similar cases were also stated to occur without any obstruction of the ducts. In the 22 cases quoted, 7 had no obstruction in the larger ducts. Moon (1929) reported three cases in a family of children with history of scarlet fever. Hæmolytic streptococci were obtained in large numbers and in pure culture from the liver in one of these cases at postmortem examination. The above as well as the cases described by Calvin and Saffro (1932), Levy, Greenstein *et al* (1939) and Harrell and Mc. Bryde (1940) and Wise and Esquill (1947) do not appear to resemble clinically the cases that we meet with in India.

Observations on the Recorded Cases

1. Aetiology

(i) *Age incidence*—Cases were divided into the following age groups and the incidence of this disease in these age groups were studied.

Years	*Male*	*Female*	*Total*
0—1	37	3	40
1—2	81	15	96
2—3	33	8	41
3—4	15	0	15
Over 4 years	8	0	8

The earliest age incidence was in an infant of two and a half months of age. It will be noticed that maximum incidence of the disease is in the age group 1-2 years.

(ii) *Sex incidence*—Only 13 per cent of our cases were females.

Sex incidence in the different age groups is shown in Fig. 1. Fig. 2 illustrates the incidence of the disease in different siblings.

It will be noticed that the maximum incidence of the disease is in the first child, the incidence in the 3rd child coming next.

(iii) *Family history*—Direct family history (*i.e.*, incidence among children of the same parentage) was obtained in 52 cases (26 per cent), and of these in seventeen instances (8·5 per cent), one after another all or nearly all the children were affected by the disease.

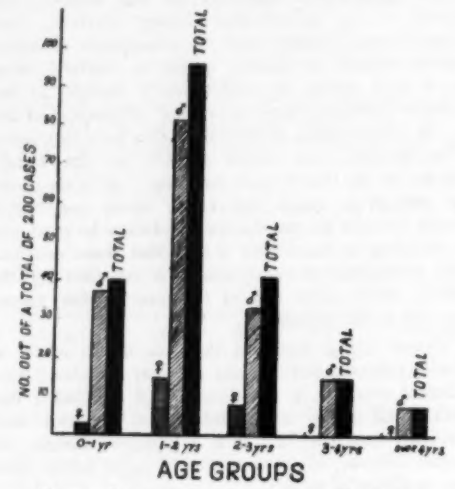

Fig. 1—Showing Sex Incidence in Different Age Groups

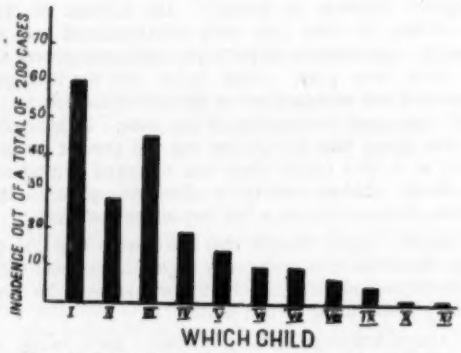

Fig. 2—Showing the Incidence of the Disease in Different Siblings

There were two instances in twins. In one instance, one of the twins were affected and the other was found healthy at the time of examination. In the other instance both the twins were affected.

Abortion—In 36 cases (18 per cent) there was history of one or more abortions.

(iv) *Religion*—

Mahomedan ... ... 6 cases (3 per cent).
Christian ... ... 2 ,, (1 per cent).
Hindu (non-vegetarian) ... 14 ,, (7 per cent).

The rest of the cases were from Jain and Hindu vegetarian families.

(v) *Class*—Almost all the cases were from the lower middle class and middle class families.

(vi) *Diet*—

Age groups	*Total cases*	*P.B.*	*A.M.*	*B. & A.*	*M.*
0—1 years	40	27	5	8	0
1—2 ,,	96	8	8	55	25
2—3 ,,	41	0	1	10	30
3—4 ,,	15	0	0	0	15
Over 4 ,,	8	0	0	0	8

N.B.—P.B. denotes pure breast feeds.

A.M. denotes artificial milk feeds (cow's, buffalo's, or goat's milk).

B. & A. denotes breast feeds supplemented with artificial milk.

M. denotes milk supplemented with solid food (cereals, lentils, vegetables and less common fruits).

It will be noticed that in the age group 1-2 years, in which the incidence of the disease was the highest, the cases (71 out of 96 *i.e.*, 74 per cent) were on purely milk diet.

2. SYMPTOMS AND SIGNS

(i) *Onset of the disease*—Almost in all the cases the disease came on very insidiously and by the time the parents noticed that something was grossly wrong with their children, and sought medical advice, the disease was fairly well advanced. Fig. 3 shows graphically the time that elapsed between the time the parents first noticed the symptoms and consulted us for medical advice. It is interesting to note that even when some parents were specially warned about the possibility of their next issue having the same disease, they did not feel the necessity for seeking medical advice earlier as they thought that their child looked quite healthy, and brought the child to us in an advanced stage of the disease.

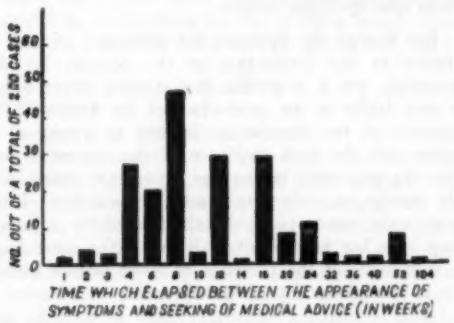
NO. OUT OF A TOTAL OF 250 CASES
60
50
40
30
20
10
0
1 2 3 4 6 8 10 12 14 16 20 24 32 36 40 52 104
TIME WHICH ELAPSED BETWEEN THE APPEARANCE OF SYMPTOMS AND SEEKING OF MEDICAL ADVICE (IN WEEKS)

FIG. 3—SHOWING THE TIME INTERVAL BETWEEN THE APPEARANCE OF SYMPTOMS AND SEEKING OF MEDICAL ADVICE

(ii) *Chief symptoms which led to the seeking of medical advice*—

Enlargement of abdomen ...	111 cases	(55·5%)
Fever (off and on, irregular) ...	76 ,,	(38 %)
Constipation	27 ,,	(13·5%)
Irregular bowels with diarrhœa off and on	16 ,,	(8 %)
Jaundice	14 ,,	(7 %)
Oedema	14 ,,	(7 %)
Weakness and wasting ...	13 ,,	(6·5%)
Cough	10 ,,	(5 %)

(iii) *Actual symptoms complained of by the parents*—

Enlargement of abdomen ...	156 cases	(78 %)
Fever (intermittent and irregular) off and on	145 ,,	(72·5%)
Bowel troubles:		
Constipation	68 ,,	(34 %)
Irregular bowels with diarrhœa off and on	64 ,,	(32 %)
Cough	111 ,,	(55·5%)
Oedema	32 ,,	(16 %)
Jaundice	30 ,,	(15 %)
Weakness and wasting ...	25 ,,	(12·5%)
Bleeding: Epistaxis ...	10 ,,	(5 %)
Hæmatemesis ...	2 ,,	(1 %)

It is apparent that the main symptoms in the order of frequency were enlargement of abdomen, fever, bowel disturbances, and cough. The high incidence of cough is interesting as an associated symptom and its significance may or may not be more than that of a chance coincidence. These children, however, are more likely to catch respiratory infections which are common in infancy.

(iv) *Physical signs*—The liver was enlarged to a varying degree in 192 *i.e.*, in 96 per cent of cases. In 8 cases associated with ascites it was not palpable. The consistency was definitely firmer than usual and in advanced cases it was hard to feel. The spleen was slightly or moderately enlarged in 92 per cent of cases, (just palpable in 36 or 18 per cent, moderately enlarged in 126 or in 63 per cent of cases and big *i.e.*, below the level of the umbilicus, in 22 or 11 per cent of cases). Icteroid tinge was noticed in 108 or 54 per cent of cases and definite jaundice was found in 77 or 38·5 per cent of cases. Visible veins over abdomen were found in 89 or 44·5 per cent of cases. The veins were carefully looked for in good light and in erect position. Sometimes they were visible in the comparatively earlier stages of the disease. Ascites was present in 59 or in 29·5 per cent of cases. It is usually a late event and is associated with portal obstruction, due to the

shrinkage of liver, anæmia, and hypoproteinæmia. Puffy face, œdema of legs and general anasarca were present in very advanced cases. Signs of rickets (bossy head, rickety rosary, thickening of epiphyses) were present in 23 or 11·5 per cent of cases. In these cases liver was more enlarged and harder than what could be accounted for by rickets alone, and treatment of rickets did not materially improve the condition of these children. Clinically these cases did not appear to be cases of gargoylism.

3. Laboratory Findings

Blood picture—Varying degrees of anæmia were present in 72 per cent of cases. The anæmia was hypochromic and only 16 per cent of cases were of the hyperchromic variety with colour index above 1. W.B.C. counts above 12,000 were present in 55 per cent of cases. The differential counts were within normal limits unless there were definitely inflammatory complications to account for the polymorphonuclear leucocytosis. Fragility of R.B.C. was tested in 22 cases to exclude acholuric jaundice and found to be withing normal limits.

Kahn test was done in 141 cases. Of these 38 were positive, one was doubtful and 102 were negative.

Stool culture—It was done in 43 cases with diarrhœa in MacConkey's and Endo's medium. There was no growth of pathogenic organisms.

Urine examination showed occasional traces of albumin and in cases with jaundice, bile salts and pigments were found.

4. Discussion

The exact causation of the disease is still uncertain. It appears that more than one of the following factors are necessary for the production of the disease.

(i) *Congenital factor*—Importance of family history has been stressed by many writers. In our series as stated above, direct family history (incidence among children of the same parentage) was obtained in 26 per cent of cases and in 8·5 per cent of cases nearly all the children one after another were affected by the disease. Such high family incidence is certainly a strong evidence of the congenital factor in the production of the disease. The writer knows of cases, where in a large joint family, the children of the same age groups were living together, eating the same food, and yet only the children of one particular member of the family got the disease and not the others. Further, one instance is known where a husband had two wives and the children by one wife were healthy whereas those by the other wife, all breastfed, died of infantile cirrhosis (observation of Col. Forsyth quoted by Narayanswamy and Tirumurti, 1939). What this congenital factor is, it is difficult to say at present. Kini's suggestion that toxins or organisms or both carried by blood through the placenta from the mother cause liver damage, and Mukherji's explanation of family incidence being due to defective quantity and defective quality of milk of nursing mother resulting from poor health, and Green-Armytage's hypothesis that deficiency of vitamins in mother's diet influences the endocrine system of the fœtus and thus predisposes to the disease, all lack scientific confirmation. We had cases where the nursing mothers were specially taken care of, given correct food, vitamins, iron, calcium and who remained in good health throughout the pregnancy and lactation periods, had children who developed cirrhosis of the liver during the lactation period. It may be that the family incidence is governed by the Mendelian law of inheritance as suggested by Prabhu (*loc. cit.*). It seems not unlikely that the Rh factor may have something to do with the congenital factor of the disease.

(ii) *Diet*—Diet is certainly a factor in the production of the disease. The high incidence of the disease amongst vegetarian Hindus of the lower middle class, whose dietary is deficient, particularly in protiens, is suggestive. Again though the disease is very uncommon in Europe, its incidence has been common when famine conditions characterised by a deficiency of protein prevailed, " and with the return of normal conditions, when meat and milk were available, the disease disappeared " (Himmsworth). Further, the disease has been described in parts of East Africa, South Africa, and the West Indies, in races where nutritional standard is poor. Again when considered from the point of view of morbid anatomy and pathological histology, it appears that the necrotic changes and the diffuse hepatic fibrosis described by Radhakrishnarao (*loc. cit.*) in his cases could be explained by deficiency of essential proteins and lipotropic factors. Experimentally conditions similar to infantile cirrhosis of liver has been produced by numerous workers in rats by keeping them on a diet deficient in essential protein and lipotropic factors.

But though the evidence for deficiency in diet as a factor in the production of the disease, is very convincing, yet it is certain that dietetic factor is not the only factor in the production of the disease. The incidence of the disease in infants as young as 2½ months old, the high incidence of the disease in our series (74 per cent) in the age group 1-2 years, who were purely on milk diet, and the incidence of the disease only among particular children of the same age group in a big joint family, all eating the same food, are suggestive of the presence of other factors besides diet, in the production of the disease.

Radhakrishnarao (*loc. cit.*) who investigated into the dietary of the children affected with the disease, found no remarkable difference between their food habits and those of normal children of the same age, caste and social position. He thought that the importance of dietetic errors in the causation of the disease was generally overstressed and very often the fact was ignored that careful enquiry would elicit the existence

of similar errors in many of the normal children of the same class.

(iii) *Toxic and infective factor*—Toxic substances and infections undoubtedly cause hepatic necrosis followed by fibrosis. The problem, however, is complicated by the fact that the action of some poisons as pointed out by Himmsworth (*loc. cit.*) is profoundly modified by nutrient factors and the action of others may conceivably be due to their causing malnutrition. That infective agents may also cause hepatic cirrhosis, directly or indirectly, has been shown by numerous workers (Weaver, 1900; Hectoen, 1901; Opie, 1910; Moon, 1929—quoted by MacMahon and Mallory, 1931) and also by Lahiri (1936). The virus of infective hepatitis may sometimes cause extensive liver damage leading to cirrhosis.

The irregular low grade pyrexia and the tendency to moderate leucocytosis which are usually present when the disease is sufficiently advanced, are suggestive of some infection which may be important in the ætiology of the disease. These may, however, be due to merely a superimposed infection. We have not been able to demonstrate so far any abnormal intestinal flora with any constancy in our series, neither have we noticed any published reports of studies in this direction. A relatively non-pathogenic organism may, however, as suggested by Opie, cause cirrhosis provided there is damage and devitalisation of the liver due to other causes. It is significant that in our series, 66 per cent of the cases had bowel symptoms (either constipation or irregular bowels with loose motions off and on). The infective and toxic aspects of infantile cirrhosis of the liver need more extensive and careful study to determine exactly their actual role in the production of the disease. Thus it seems that there is good deal of truth in the statement made by Sen about 60 years ago, " that some constitutional predisposition or dyscrasia is inherited by the children which under any of the least exciting causes gives rise to the disease."

V. Treatment

Studied from the therapeutic point of view, it was not possible to arrive at a definite conclusion regarding the method of treatment which was most useful in these cases. Most of the patients were brought in our hospital in a fairly advanced stage of the disease and the parents could rarely be persuaded to keep their children sufficiently long in the hospital to enable one to evaluate the efficacy of a certain method of treatment. Inspite of repeated attempts it was not possible to follow up these cases subsequently.

My impressions from my private series of cases are as follows:—

(i) Some cases recover when given a simple general line of treatment *e.g.*, diet of skimmed milk, fruit juice (and in older children, in addition, cereals, lentils, boiled mashed vegetables without ghee, oil or spices) and vitamins A, B, C, D, iron, liver extract and symptomatic treatment.

There could be no doubt that these cases were of genuine cirrhosis of the liver (they had positive family history, had hard enlarged liver, palpable spleen and had visible veins over abdomen). Other cases and a larger number, on this method of treatment gradually get worse and die. It is interesting to note that sometimes owing to financial or other reasons the treatment prescribed could not be followed for any length of time and yet some of these cases recovered.

(ii) Medicines like salol, kalmegh, zinc oxide and the numerous proprietary remedies widely advertised in the lay press are of very doubtful value. If one critically analyses the cases, one is not at all convinced about their efficacy. Apparent occasional recoveries may be explained by other factors, namely diet and general measures.

(iii) Since the time substances like methionine, choline, protein and casein hydrolysates have become freely available, the recovery rate seems to be definitely higher, though a number of cases still continue to go downhill and die in spite of a generous supplement of these substances. It is not difficult to understand why some cases apparently recover without any special treatment while others apparently at the same stage of the disease, as far as one can judge clinically, inspite of all possible remedies gradually become worse and ultimately die. It is not easy to judge in a given case how far the congenital factor is responsible. It is conceivable that if the inherited defect in the liver is of severe degree, correction of dietetic errors, administration of lipotropic factors, vitamins etc., and control of infective processes may not be sufficient to avert the progress of the disease and the ultimate fatal issue.

In the present stage of our knowledge of the pathogenesis of the disease, the following is suggested as a rational approach to the problem of the treatment of the disease:

As the congenital factor of the disease is beyond the scope of practical therapeutics, careful attention should be paid to the other two factors, namely, dietetic and the possible infective or toxic factor.

Diet—In very young infants breast feed should be continued unless there be any special reason for putting the child on artificial milk; older infants may have skimmed milk, skimmed buttermilk, fruit juice, and depending on the age of the child, cereals, lentils, vegetable purees, minced chicken, fat-free minced meat, steamed or boiled fish. Food should not be spiced and fats should not be used for cooking food for active and advanced cases.

Medicines—

(i) Essential amino acids, *e.g.*, protein hydrolysates, casein hydrolysates and lipotropic factors like methionine and choline should be given in therapeutic amounts.

(*ii*) As most of these cases have hypochromic anæmia iron is indicated.

(*iii*) As a state of subnutrition exists in most of these cases, possible multiple deficiency in vitamins should be remedied by the administration of vitamins A, B, C, D, and particularly vitamin B complex and vitamin E. Riboflavin deficiency is said to lead to gross fatty infiltration of liver and pyridoxin is reputed to exert a lipotropic action. Inositol is also supposed to have a lipotropic action. Till the action of these substances are more accurately known it would be advisable to use a preparation of vitamin B complex containing these substances in therapeutic amounts. Since tocopherol can prevent hepatic necrosis in experimental animals living on a necrogenic diet and since relatively little tocopherol is provided by maternal milk, administration of tocopherol in addition to other vitamins is indicated.

(*iv*) Crude liver extract and vitamin B_{12}—These are indicated in cases associated with hyperchromic anæmia. Routine use of these substances in view of the supposed beneficial action in cirrhosis of the liver, is not desirable if the child is otherwise improving, as their use may not be of much value and involves unnecessary painful injections to a small child.

(*v*) Antibiotics—In view of the possible part played by organisms of *B. coli* group and streptococci, it would be rational to use antibiotics like aureomycin and terramycin particulary in cases having irregular rise of temperature without any other apparent cause. Accurate and controlled observation is, however, necessary before the real value of these antibiotics in infantile cirrhosis can be assessed.

SUMMARY

1. Infantile cirrhosis is not uncommon in Madhya Bharat.

2. The literature on the subject has been reviewed.

3. Statistical data regarding the ætiology and clinical features of the disease have been described.

4. The ætiology and the treatment of the disease have been discussed.

ACKNOWLEDGMENT

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PRIMARY INTESTINAL TUBERCULOSIS

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This note is based on the histopathological study of the material received from various units of our hospitals. This study has brought to our attention certain gaps and discrepancies in our knowledge of this subject and has focussed our attention on the many difficulties in the diagnosis of "Primary Intestinal Tuberculosis."

Review of the literature on this subject as published in India does not seem to be very informative. The contributions of Tribedi (1941) and Ukil (1942) do not seem to throw much light on this subject, as both the studies were not accompanied by comprehensive histopathological data. A few excellent reviews on this subject have appeared recently abroad. Shields Warren (1948) studied the problem of regional ileitis and Hoon *et al* (1950) reviewed the material of ileocolitis at the Mayo Clinic.

During the years 1949, 1950 and 1951 a total of 26 specimens consisting of resected loops of bowels comprising of the terminal ileum, cæcum and proximal colon were received in the department of pathology for histopathological examination.

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in the umbilical region or round about the right iliac fossa; loss of appetite; constipation and/or diarrhœa. There was no cough. A low irregular rise of evening temperature was noted in some cases. Loss of weight, though complained of by some was not very definite. Duration of symptoms was variable being as short as 6 months and in some cases as long as 6 years.

The physical signs in all these cases were more or less of the same order. They all showed a diffuse lump in the right iliac fossa which was movable in some and fixed in other cases. The mass was tender on pressure. The mesenteric glands were felt enlarged in some cases. The general feel of the abdomen was described as " doughy." Liver and spleen were not palpable.

Respiratory and cardiovascular systems were reported as normal. X-ray of the chest when done, showed no evidence of active pulmonary tuberculosis. Laboratory examinations were negative except for few red cells and pus cells in the stool of some cases. Blood examination revealed a mild degree of anæmia. A clinical diagnosis in all these cases was one of " intestinal tuberculosis " or " cæcal tuberculosis " or " ileocæcal tuberculosis."

Histopathological examination of all these 26 resected loops of bowel revealed 6 of them to be neoplastic conditions of the intestines (primary lymphosarcoma of the ileum 2, carcinoma of ileum and cæcum 4).

The remaining cases form the subject matter of these observations. The age group of these patients varied between 16 to 40 years, 75 per cent of them being below 30 years, the average age being 25 years. Twelve belonged to the female sex and six were males.

The gross examination of these specimens showed some striking similarity with minor differences. The lesions were confined in all cases to the terminal ileum and cæcum. They all presented a gross picture of chronic granulomatous mass with thickening of the proximal ileum. In some, the mucous membrane was ulcerated and necrotic, in others small polypoid projections on the mucous surface were visible. The degree of stenosis at the ileocæcal junction, though always present, was variable and so was the thickening and fibrosis of the adjacent bowel wall. Search for the presence of " tubercles " on the serosal surface showed in only four, small nodules of yellowish white colour on the bowel wall. In some specimens a few enlarged lymph glands could be seen along the border of the intestinal loops. These glands were firm and the cut surface showed no caseation.

The histopathological studies of the affected bowel wall and the lymph glands were made. The common pattern in all these specimens had been one of diffuse granulomatous process consisting of the crowding of the various coats of intestinal walls with lymphocytes, plasma cells, monocytes and eosinophils and a variable degree of fibroblastic proliferation and fibrosis. Necrosis of the mucous membrane was variable but was always present. In all these specimens a focal collection of cells presenting the appearance of a " tubercle " with foreign body type of giant cells was noticed. The number of these " tubercles " was variable; in some specimens they were in large numbers, in others scanty. The most striking histological feature, however, has been the absence of caseation—pink-stained homogenous area—either in any coat of the bowel wall or in close proximity of the cell aggregations—" tubercles " or in the lymph glands. A careful study of all these sections failed to reveal any areas of caseation. Only in one case there was a suggestion of such areas in the bowel wall and corresponding lymph nodes. We stained all the sections with silver impregnation and demonstrated the presence of reticulin network or strands of collagen in the centre of these " tubercles." Microscopic sections stained for acid-fast bacilli were consistently negative.

The problem, therefore, which has emerged out of this study is as to the ultimate nature of these lesions. Ordinarily, the histopathological appearances may be considered by some to be sufficiently indicative of a tuberculous process. We have no doubt, such pathological appearances, backed by clinical diagnosis have been responsible in the past for a diagnosis of " primary intestinal tuberculosis." In view of the absence of caseation in any of these lesions and the failure to demonstrate acid-fast bacilli, we have reasons to entertain doubts about the tuberculous nature of these conditions. Presuming that they are atypical reaction to primary infection of intestine with tubercle bacilli—human or bovine, it is difficult to believe that " caseation," which is a recognised lesion of tissue reaction to tubercle bacilli should be so consistently absent in this material.

Shields Warren (1948) on the basis of examination of 120 specimens which showed more or less the same type of gross appearances and histological features as in our material, came to the conclusion that " 'cicatrizing enteritis ' has absorbed almost all the cases of once so-called hyperplastic tuberculosis of intestines and that this term is now practically obsolete." In his opinion there is every justification in accepting " cicatrizing enteritis " as a separate pathologic entity. We feel, however, that the diagnosis of " cicatrizing enteritis " does not solve the problem. It only means the introduction of a new name to describe the non-specific nature of a chronic inflammatory process in the region of the terminal ileum and cæcum. This does not attempt to clarify the basic nature of the lesion. Hoon *et al* (*loc. cit.*) in a review of the material available at the Mayo Clinic from 1921-1946, have brought out the possibility of the existence of a third type of pathological lesion in the ileocolic region in addition to tuberculous ileocolitis and " cicatrizing enteritis " (Crohn's disease). They suggest that some of these, histologically indistinguishable lesions, may be cases of " isolated intestinal sarcoidosis " or what they call " non-caseous tuberculated enterocolitis."

In view of the non-specific nature of histological lesions in this material we have studied and the views of Shields Warren and Hoon, we feel that the problem requires further eleborate investigations before labelling them as tuberculous. With this object in view, a project has been taken up in our institution. The resected loops of the bowel wall and the lymph nodes are being subjected to a bacteriological examination by culture on a number of special media, and inoculated into guinea pigs, in order to prove or disprove definitely the presence or absence of tubercle bacilli in these lesions. We are also planning to type the tubercle bacilli, if isolated, into human and bovine. Unless large scale investigations of this type are undertaken, it is not advisable to label these cases as " primary intestinal tuberculosis." The problem in our opinion needs to be studied with an open mind.

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GARLIC JUICE IN THE TREATMENT OF ULCERS*

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In a previous article Gupta (1952) has observed that garlic juice has bacteriostatic properties for a number of pathogenic organisms. Few attempts, however, have been made on its therapeutic applications. Chopra (1933) mentions that the application of garlic juice diluted 3 or 4 times with water to infected wounds resulted in diminution in pus discharge and relief of pain. Tokin (1943) mentions that it has been used as wound dressing in Russia. The present study was undertaken with a view to observe the effect of crude freshly obtained " garlic juice " on ulcers.

* The work was carried out with a grant from U.P. Government in the Department of Pathology, Medical College, Lucknow and in Surgical Out-Patients Department of Gandhi Memorial and Associated Hospital, Balrampur Hospital and King's English Hospital, Lucknow.

Type of Bacteria in Ulcers

A bacteriological study of the chronic wounds, ulcers, and sinuses commonly seen in surgical out-patients was undertaken. Swabs were taken from the surface of 50 ulcers. Smears were made and cultures done on blood agar medium and incubated aerobically at 37°. The following bacteria were identified by these methods:—

Organisms	*No. of cases*
Staphylococcus aureus	31
Staphylococcus albus	8
Streptococcus hæmolyticus	20
Diphtheroids	10
Bacterium coli	5
Proteus vulgaris	5
Pseudomonas pyocyaneus	1
Streptococcus viridans	1
Fusiform bacilli	1

Sensitivity of Organisms to Garlic Juice

An attempt was made to devise a simple test to determine if the organisms were sensitive to the action of garlic juice. For this purpose the organisms were inoculated in 5 per cent garlic juice in broth or 10 per cent garlic juice in blood agar plates. Parallel broth and blcod culture plates without the garlic juice were also set up as controls. Most of the organisms which were isolated from these ulcers failed to grow in the media containing garlic juice and were considered to be sensitive to garlic juice.

Bacteriostatic Action of other Dressing Substances

In order to have a comparative idea of the bacteriostatic activity of the common dressing solution eusol, doubling dilutions of Seitz filtered garlic juice and eusol were made with peptone water. All the tubes including a control were inoculated with a loopful of culture of staphylococcus aureus and Bacterium coli, incubated at 37° for 24 hours and the highest dilution which did not show any turbidity was taken to be the bacteriostatic strength.

The results were as follows:—

Garlic Juice %	*Staphylo-coccus aureus*	*Bacterium coli*	*Eusol %*	*Staphylo-coccus aureus*	*Bacterium coli*
50	–	–	50	–	–
25	–	–	25	+	[+]
12	–	–	12	+	[+]
6	–	–	6	+	[+]
3	–	–	3	+	[+]
1·5	–	+	1·5	+	+
0·8	+	+	0·8	+	+

+ Growth. – No growth.

From the above experiments it was obvious that garlic juice possessed far greater bacteriostatic properties than eusol for staphylococci and coliform organisms.

Treatment of Ulcers with Garlic Juice

The treatment of ulcers was started in the form of dressings. The ulcers were dressed with 15 per cent garlic juice once a day after cleaning them with tap water. Applications of garlic juice removed the pus in most cases in a few days leading to the appearance of healthy granulation tissue and later the skin started growing from the edges. In addition to the clearing and healing effects, garlic applications showed definite pain relieving effect in most of the cases. Garlic juice dressing was tried in 335 cases. Of these 169 (about 50 per cent) stopped coming to the hospital after one or two dressings while 40 cases stopped the treatment when they were partially healed. 123 cases persisted in treatment and of these 111 were completely healed, while treatment had to be changed in 12 cases. The strength of garlic juice used in these cases was as follows:—

Garlic juice strength	*No. of cases*
50%	10
25%	102
15%	30
10%	24

In 21 cases of ulcers attending the orthopædic outdoor it was observed that pain was less in 20 cases, it increased in one. Pus discharge less in all cases.

Effect on Ulcers

Observations on cases which did not respond to 15 per cent suggested that higher strengths should be used. Gradually the strength of garlic juice was increased and it was found that in cases with excessive pus and slough 50 per cent garlic juice should be used and as soon as the wound cleared up and granulation tissue appeared, the strengths should be reduced to 25 per cent. When the skin started growing a 20 per cent or 10 per cent garlic juice suspension in "til oil" was used in order to avoid sticking of dressings and bleeding from granulation tissue. 10 per cent garlic juice in "til oil" was found to be very useful in two cases of extensive burns.

Rate of Healing

In order to have an idea of the rate of healing of common ulcers with garlic juice treatment, the area of ulcers was calculated in 36 cases by measuring it in two diameters. The number of days required for healing were added and the average healing rate per day was calculated by dividing the total area by the number of days. For comparison, similar observations were made on 9 cases dressed with eusol and the results were as follows:—

Dressing with	*Healing per day in sq. inches*	*No. of cases*
Garlic Juice	0·095	36
Eusol	0·082	9

The above observations show that the healing rate was practically the same with both garlic juice and eusol.

Resistant Cases

The cases in which dressings with garlic juice were not effective were of the following types:—

1. Cases with maggots. It was found that maggots could live for hours when put in pure garlic juice.

2. Ulcers with sinuses.

3. Ulcers associated with tubercular or pyogenic bone diseases.

4. One case of Naga sore with fusiform bacilli was not affected by 15, 30 and 40 per cent of garlic juice but when 50 per cent garlic juice was used, it rapidly healed up.

Conclusions

The above observations show that garlic juice is definitely useful in the treatment of wounds and ulcers and aseptically collected fresh garlic juice should have a place in the dressing of ulcers. It is a simple, cheap and easily available drug for this purpose. Only fresh garlic juice is useful, as it loses its antibacterial activity on heating or keeping for a few weeks (Gupta 1952). It cannot be bottled on commercial basis.

Acknowledgment

In the end we thank the U. P. Government for the research grant and for lending the services of Dr. Bhartendu Agarwal of Provincial Medical Service for help in the research work. We thank the colleagues and in particular Mr. Swaran Singh, the Research Assistant, for their help in this work.

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PRACTITIONERS' CORNER

IMMEDIATE EFFECT OF DEWORMING IN PATIENTS WITH PEPTIC-ULCER-LIKE SYNDROME

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Yenikomshian and Shehadi (1943) reported on patients with clinical histories suggestive of duodenal ulcer and with ova of ankylostoma duodenale in the fæces. All the signs and symptoms were rapidly eliminated, the epigastric pain was relieved, after an anthelmintic was given.

Khalil Bey and Salah (1935) reported symptoms like those of duodenal ulcer but with duodenal cap present on skiagram, they found that the symptoms and occult blood were no longer present after deworming.

A group of patients consulted for pain in epigastric region for a period varying from three months to two years. Pain was at times aggravated 3 to 4 hours after food. They had occasional nausea and vomiting. The patients complained of alternate constipation and diarrhœa. The stool examination showed hookworm infection. They were straightway given a course of tetrachloroethylene (4 ml. in capsules) provided the blood conditions permitted and the effect of the anthelmintic on the syndrome was noted.

15 patients, all of whom complained of peptic-ulcer-like syndrome, were followed up. 11 out of these 15 patients gave a positive occult blood test.

TABLE SHOWING IMMEDIATE EFFECT OF DEWORMING IN A SERIES OF PATIENTS OF HOOKWORM HAVING A PEPTIC-ULCER-LIKE SYNDROME

No. of cases	*No. in whom symptoms disappeared*	*Percentage*	*Positive occult blood test*	
			Before treatment	After treatment
15	13	86·6	11	2

The table shows that in 86·6 per cent of the patients the symptoms disappeared immediately after deworming. In fact, when the patients returned in the evening to report, they informed with a smile that the purgative had cleared away all pain in the abdomen.

It is also interesting to note that 11 out of those 15 patients had occult blood in their stool and only in two patients occult blood persisted after deworming.

Apparently, the hookworms set up a mild duodenitis leading to peptic-ulcer-like syndrome. The diagnosis is made all the more difficult as occult blood is present in the majority of these cases. The stool examination, however, shows hookworm ova and if a course of anthelmintic is prescribed the symptoms disappear in 86·6 per cent of the cases pointing to the duodenitis set up by the hookworms.

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HYPERTENSION*

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According to William Evans (1948), blood pressure should not be regarded as high unless the systolic reading is 180 or over, and diastolic reading 110 or over, for at least three consecutive occasions, and when there is also present a clinical, cardiographic and cardioscopic hypertrophy of the left ventricle. According to Frederic Price, in hypertension the systolic and diastolic B.P. readings are persistently above certain levels whatever the cause, the levels generally agreed to being 160 systolic and 95 diastolic in adult males and 170 and 100 in elderly persons.

PREDISPOSING AND AETIOLOGICAL FACTORS

(*i*) 90 per cent of cases are over 40 years of age when symptoms first occur; (*ii*) Greater stress and strain of modern life is believed to be responsible for an incidence of the disease. It is far more common amongst the Europeans than in Indians and is rare in African Negroes; (*iii*) Heredity certainly plays an important part in the incidence of this disease; (*iv*) Both sexes are affected; females more than the males, and in the former age incidence is also higher which is attributed to the inhibiting action of ovarian secretions; (*v*) The obese have an arterial B.P. 10 mm. of Hg. higher, and very thin people have about 12 mm. lower than those of normal build; (*vi*) A lot of fuss has been made about high protein diet, but experimental results of high protein diet are very conflicting and do not support the view that hyperproteinæmia leads to hypertension; (*vii*) Hypercholesterolæmia is present in atherosclerosis and atherosclerosis is often a cause of raised B.P. but increased cholesterol content of the blood *per se* is not the cause of essential hypertension and blood cholesterol is never found to be increased in it; (*viii*) Diabetes mellitus leads to hypertension in some cases; (*ix*) Although some endocrine disorders are associated with hypertension, very few cases of essential hypertension show any endocrine changes either chemically or post-mortem.

The actual cause of essential hypertension is, therefore, indefinite. Goldbladt in 1928 first successfully produced experimental hypertension by clamping the renal artery of dogs and monkeys, but his experiments are not applicable to man. According to Pickering, Prinzmetal and Wilson, the causal agent is probably chemical, and this chemical agent is responsible for the production of hypertension by direct action on the vascular bed.

Nature of chemical agent—Tigerstedt and Bergmann in 1898 mentioned a sustance known as renin

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having a powerful and prolonged pressor action, and this can be extracted from normal kidneys. Renin, unlike other powerful pressor substances like pituitrin and adrenaline, does not decrease the peripheral blood flow, *i.e.*, the hypertension produced is similar to that of primary hypertension. Renin is found in the kidneys. Kidneys take some part in the elimination of renin; but it is mainly destroyed by the tissues. Renin is not concerned with the maintenance of blood pressure but with its regulation. The pressor action of renin is produced through the formation of hypertensin (angiotonin) formed by its action on another blood globulin enzyme hypertensinogen which is manufactured in the liver, *i.e.*

Renin and Hypertensinogen → Hypertensin (pressor compound).

Unlike renin, hypertensin is heat-stable and soluble in various organic solvents. This pressor substance, hypertensin, acts on the blood vessels. The normal kidney tissue also secretes another enzyme called hypertensinase which destroys the activity of hypertensin. In essential hypertension this particular enzyme equilibrium appears to be disturbed.

Recently, Hans Selye has put up another theory of "general adaptation syndrome" and says that hypertension is a disease of adaptation due to hyperfunctional endocrine disturbances, and says further that diseases like hypertension, nephrosclerosis and rheumatic diseases may represent by-products of the endocrine reactions which are at play in the general adaptation syndrome.

Classification

Classification of hypertensin is best made on ætiological basis.

Aetiologically hypertension is—

1.	Essential—	(*i*) Benign. (*ii*) Malignant.
2.	Due to cardiovascular disease—	(*i*) Co-arctation of the aorta. (*ii*) Aortic incompetence. (*iii*) Atherosclerosis.
3.	Due to renal disease—	(*i*) Acute nephritis. (*ii*) Chronic nephritis. (*iii*) Pyelonephritis. (*iv*) Polycystic kidney. (*v*) Abiotrophic kidney. (*vi*) Toxæmia of pregnancy.
4.	Due to endocrine disease involving—	(*i*) Pituitary. (*ii*) Adrenal. (*iii*) Thyroid. (*iv*) Thymus. (*v*) Ovary.
5.	Due to metabolic diseases—	(*i*) Diabetes mellitus. (*ii*) Gout.
6.	Due to blood diseases—	(*i*) Polycythæmia vera. (*ii*) Polycythæmia hypertonica.

(1) Essential Hypertension: *Benign*—It is also called simple hypertension, primary hypertension and red hypertension. The majority of cases fall into this group and are between 50-60 years of age. According to Evans (1948) the symptoms put down as headache, giddiness etc., are in reality symptoms of neurosis and not hypertension. The real symptoms are epistaxis, hæmoptysis, hæmatemesis and other phenomena of rupture of a blood vessel, cerebral thrombosis, cardiac infarction, hæmorrhage in the retina or other ocular catastrophe giving dimness of vision, cardiac asthma etc. Full bounding pulse and raised B.P. are there, carotid pulse due to kinking produced by a rising aortic arch is a common physical sign. Apex beat is displaced. On auscultation there is a loud second sound in the mitral and the aortic areas. A systolic murmur due to hypertension is heard only when there is a very great enlargement of the left ventricle and according to Evans (*loc. cit.*) this murmur is muscular in origin. It is heard in middiastole. E.C.G. shows left axis deviation, lowering of ST segment and there may be inversion of T waves. Cardioscopy shows enlargement of the left ventricle of the heart, elongation of aorta, and pulmonary congestion.

Examination of eye fields may show normal fundi or at the most arteriosclerotic changes in the vessels, without any papillœdema. hæmorrhage and exudate, *i.e.*, Grade 1 and 2 of Wagener and Keith.

Malignant Hypertension—It has also been named by Volhard as pale or white hypertension. The course of this variety of hypertension is much accelerated and is crowded with serious symptoms. Here one finds pathologically, arteriolitis and arteriolonecrosis, in contrast to diffuse arterial fibrosis which is characteristic of benign hypertension and is of slow evolution.

Clinically, it is seen more commonly in young people, females more than the males, and the commonest symptoms are morning headache, cardiac asthma and failing vision. There may be lassitude, anorexia, loss of weight, and anæmia. Blood pressure readings are much higher than in benign hypertension, the diastolic going upto 120 or higher and the systolic remaining more than 200 mm. of Hg. Examination of eye fields shows constantly marked changes in the fundi and the retinopathy is usually early. There is marked arterial constriction, narrow attenuated arteries —tortuous and silver-wire in appearance; full, engorged veins which show marked nipping at their crossing with the arteries, hyperæmia, massive papillœdema, many flame-shaped superficial hæmorrhages and soft cotton-wool exudative patches *i.e.*, Grade 3 and 4 of Wagener and Keith. This picture of retinitis is of great prognostic and diagnostic significance and often heralds a death warrant, for very few of these patients live for two years or more.

(2) Hypertension in Cardiovascular Diseases: (*a*) In *atherosclerosis* the B.P. is only raised if it happens to complicate cardiovascular hypertrophy. Here, too, besides irregular, thickened and tortuous

arteries pulsating under the skin, a sustained B.P. and cardiac hypertrophy with a marked systolic murmur in the apical area and at the base, we find marked changes in the fundus oculi *i.e.*, highly tortuous, irregular, copper-wire or silver-wire arteries, sheathing, and nipping of the veins, and small bright, hard exudate, spots in contrast to soft woolly patches of malignant hypertension. The hæmorrhages may or may not be present and are usually small and scattered. There is no papillœdema and the optic disc for all purposes is normal. The condition is usually bilateral. Again, unlike the retinopathy of malignant hypertension this retinopathy is not of very definite prognostic significance.

(*b*) In *aortic incompetence* the systolic reading is usually high and the diastolic low, which give a high pulse pressure. The systolic pressure in the femoral artery is higher than in the brachial.

(*c*) In *co-arctation of the aorta* there is an inequality of B.P. and pulse volume in the upper and lower extremities. The systolic pressure in the arms is very high, 200 mm. of Hg. or more, the B.P. in the femoral artery is very low, 100 mm. or less, and the femoral pulse is small. The pressure in both arms is generally equal. According to Maurice Campbell, high B.P. without reason, striking pulsation in the neck, signs of aortic incompetence and a systolic murmur, absent or very small femoral pulse, point to a clinical diagnosis of co-arctation of the aorta. Most patients with co-arctation are, indeed, very well physically. The diagnosis can be confirmed radiologically, when x-ray examination will show cardiac (left heart) enlargement, absence of aortic knuckle and notching and erosion of the ribs by the dilated intercostal arteries.

(3 RENAL HYPERTENSION: Only 12 per cent of all cases of hypertension fall into this group. Here we find that the hypertension is progressive and arises as a complication of the primary kidney disease which causes renal destruction and impairment of kidney function.

In *acute* (*type I*) *nephritis* the patient, usually a young adult with an ætiological background of acute infection of the upper respiratory passages, suddenly develops hæmaturia, oliguria, œdema, hypoproteinæmia and arterial hypertension, which is moderate *i.e.*, rarely higher than 180 systolic and 120 diastolic. The blood pressure comes to normal in 80-84 per cent cases which make a complete recovery in this disease.

In *chronic* (*type 2*) *nephritis* there is presence of dropsy, anæmia and cylindruria besides hypertension. The renal retinopathy here is practically the same as described in malignant hypertension and so is its diagnostic and prognostic significance.

Pyelonephritis (pyelitis) resembles chronic glomerulonephritis in its ultimate effects. According to Weiss and Parker, it is responsible for 15 to 20 per cent of cases of malignant hypertension; and according to them hypertension in this disease is independent of renal function.

Polycystic disease of the kidneys presents itself generally as a bilateral condition with large palpable kidneys, besides high B.P. and cardiac hypertrophy. There is dull aching pain in the loins, general malaise and recurrent hæmaturia.

Toxæmia of pregnancy—Some cases of pregnancy show hypertension, presumably due to toxæmia of pregnancy. In some of these cases after three or four confinements a retinopathy occurs by about the 6th month of pregnancy, and the condition simulates malignant hypertension.

(4) ENDOCRINAL HYPERTENSION: *Basophil adenoma of the pituitary* first described by Cushing in 1932 causes hirsuties, obesity, purplish cutaneous striæ and a high blood pressure, and early amenorrhœa in females and impotence in males. This is known as Cushing's disease, to distinguish it from Cushing's syndrome in which the clinical features are the same but the lesion is in the adrenals. In both Cushing's disease and Cushing's syndrome the obesity (" buffalo-type ") is characterised by a pad of fat over the clavicles and upper dorsal spines, a fat face, trunk and abdomen, and normal-sized limbs. There is, also, present osteoporosis, and muscular weakness. This pituitary basophil adenoma is generally too small to give rise to any pressure symptoms in the eyes *i.e.*, there is no papillœdema, optic atrophy or restriction of visual fields.

Hypertension may also be present in a case of *acromegaly*—the disease which produces large spade-like hands, prognathism and macrosplanchnia. An acromegalic patient may erroneously be treated for enlarged liver or for high B.P. without recognising the basic condition.

Pheochromocytoma is the adrenal medullary tumour which causes hypertension. It is also called chromaffinoma or paraganglioma. This tumour is rich in adrenaline and noradrenaline, and it is believed that noradrenaline causes asymptomatic paroxysmal hypertension while attacks of tachycardia and marked subjective symptoms follow the release of adrenaline. The picture of a patient seen in an attack is unforgettable. The attack manifests itself in paroxysms of marked shivering, sweating, pallor, palpitation and dyspnœa, and in some, tremors, nausea, vomiting and cyanosis. The B.P. during the attack is high *i.e.*, upto about 200 mm. Hg. or so and normal in between the attacks. Recently, cases of persistent hypertension even simulating the malignant type have also been described. The condition is diagnosed by the typical attack, perirenal insufflation of air (about 450-500 c.c.) and by retrograde pyelogram; and by benzodioxane and histamine tests. Benzodioxane 0·25 mgm. per kilo of body weight is given intravenously slowly and a fall in B.P. of some 60 mm. of Hg. is noticed in

pheochromocytoma, the fall lasting for some 15 minutes. In essential hypertension very transient minor fall in B.P. is noticed. Histamine test done by giving 0·05 mgm. of histamine in 0·5 c.c. of normal saline intravenously is done to induce the characteristic paroxysmal attack with a great rise of B.P.

Thyrogenic hypertension, though described by Sir John Parkinson and Clifford Hoyle is still debatable. In thyrotoxicosis the systolic pressure readings are generally higher and the diastolic normal or low, thus giving a high pulse pressure. According to Parkinson, patients with hyperthyroidism have a tendency to hypertension.

Thymic hypertension is known and recognised, but very little is known of *ovarian hypertension*.

5. METABOLIC HYPERTENSION: Diabetes mellitis and gout predispose to atherosclerosis which may cause consequent rise of B.P.

6. HYPERTENSION ON ACCOUNT OF BLOOD DISEASES: In polycythæmia vera (Osler-Vaquez disease) headache, vertigo, and paræsthesia, cyanosis, red shotty eyeballs, high blood-pressure readings, enlarged spleen and a blood picture showing increased R.B.C. *i.e.*, about 8-10 million per c.mm. and a Hb. of 150-200 per cent are characteristic. All these symptoms are due to increased viscosity of blood. In polycythæmia hypertonica (or Geisböck's disease) there is polycythæmia and hypertension, albuminuria, left ventricular hypertrophy and usual sequelæ of high B.P. but no splenic enlargement.

SEQUELAE AND PROGNOSIS

Benign hypertension usually runs a slow and prolonged course, fifteen to twenty years and more after the development of symptoms. More than 50 per cent from cardiac causes (*i.e.*, congestive heart failure and left ventricular failure), quite a large number from cerebral hæmorrhage and a few from uræmia. In malignant hypertension uræmia tops the list, and cardiac and cerebral hæmorrhage come next in order as the cause of death.

Persistent high B.P. in essential hypertension gives " hypertensive crises "—the so-called attacks of hypertensive encephalopathy. The attacks are precipitated by a further sudden rise in the already high pressure leading to angiospasm, cerebral anæmia and cerebral œdema, and characterised by extremely severe headache, sickness, drowsiness and semicoma, or convulsions. Hemiparesis is commonly found in the attack, hemianopia or other indication of local cerebral lesion being less common. Eye fields show the usual hypertensive changes with papilloedema which signifies that cerebral œdema is also present. The crisis is short-lived—lasting from a few hours to 2 to 3 days and ends in recovery, but it has a tendency to recur. The end in these patients results from cerebral hæmorrhage.

TREATMENT

Treatment of cases of essential hypertension should, first of all, aim at avoiding all " pressure phobia " in the patients, and adequate rest of at least 8-10 hours daily.

Diet—In the past, the hypertensives were generally put on restricted diet. As has already been pointed out in the beginning, experimental evidence has failed to prove that high protein or high fat diet leads to hypertension. It is not necessary, therefore, to ask the patient to cut down these articles from his food. ' Moderation ' should be the watchword in everything including alcoholic drinks. With regard to tobacco and smoking it should be remembered that nicotine is a vasconstrictor, and as such elevator of blood pressure, therefore, smoking should be stopped or cut down to the minimum. Vitamin A in large doses has been advocated by some but it is of no value. The therapeutic value of rice diet has not proved of merit especially in Indian patients.

Drugs—For restlessness and insominia bromides and barbiturates are the drugs of choice. In severe headache and in hypertensive encepalopathy hypertonic solutions like Mag. sulph 2 gm. in 10 per cent solution or 25 c.c. or 50 c.c. of 20-25 per cent glucose should be given intravenously. Venesection and lumbar puncture are also of value in hypertensive crises and headache. Venesection is also useful in plethorics and those who show great distension of right heart. At least a pint of blood should be withdrawn and this can be repeated at intervals.

Dramamine (β-dimethyl amino ethyl ether 8—chloro-theophyllinate) a labyrinthine depressant first used by the Americans in 1948 in sea and air sickness, has been found useful in vertigo and vestibular disturbances complicating arteriosclerotic and hypertensive cardiovascular disease by Goldman *et al* (1951), in doses of 25 to 100 mgm. four times daily, and there is no recurrence of symptoms after the discontinuance of the drug. Thiocyanates are effective in reducing B.P. though their mode of action is not known. Severe and intractable headache is the most important indication of their use; and the principal drug used has been potassium thiocyanate 6-9 grains a day and then half the dose after first 5 or 6 days, this gives a concentration of about 10 mgm. per 100 c.c. of blood which is considered to be the effective blood level. The blood level of 15 mgm. per 100 c.c. of blood should never be exceeded and the use of the drug should be controlled by the estimation of blood thiocyanate weekly. 50 per cent cases of essential hypertension respond to it, but the B.P. rises again when the drug is withheld. Its toxic effects are very severe and include fatigue, headache, vertigo, mental confusion, nausea and vomiting, skin rashes, anæmia, jaundice and death. Its contra-indications are atherosclerosis, renal disease and old age.

Rauwolfia serpentina alkaloids were found by Chopra *et al* (1933) to possess sedative, hypnotic and

hypertension lowering properties. They found that the alkaloid serpentine in doses of 1 mgm. per kilogram of the body weight would cause a fall of 30 to 40 mm. of Hg. Bhatia and Kapur (1944) describe very favourable results with Rauwolfia Serpentina. Vakil (1949) remarks that this drug, has a definite place in the treatment of cases of high B.P. Very recently, Mazumdar and Mukherjee (1951) watched its effects on 12 patients and found that 7 showed moderate lowering of B.P., in 1 it was slight and in 3 the readings remained the same. Personally, I have found that it gives good results in cases of benign hypertension, reducing the B.P. by about 40-50 mm. of Hg. but is of little use in cases of malignant hypertension.

Salts of pentamethonium and hexamethonium possessing the property of blocking the transmission of nervous impulses through autonomic ganglia and thereby interrupting the vasomotor tone were first described by Paton and Zaimis (1948a and 1948b), and their clinical trials in the treatment of hypertensive disease done by various workers like Arnold and Rosenheim (1949), Burt and Graham (1950), Restall and Smirk (1950) and Turner (1950). The drugs were first given parenterally and was later found that they could also be given by mouth though much larger doses were needed by this route to produce the same effects on account of uncertainty of absorption this way and acquired tolerance to oral dose. The way how they have been recommended to be given is: make a thorough examination of the patient including his cardiac and renal function, then do the sedation test (by giving amytal gr. iii hourly for three hours and noting the B.P.) and then give hexamethonium bromide subcutaneously or intramuscularly in the initial dose of 20 mgm., the patient lying in a propped up position in the bed. A fall in B.P. is immediately noticed and may be followed by dizziness. Increase or decrease the second dose according to response of the first dose, and repeat the injections twice daily. Once a satisfactory control is obtained, the drug can then be given by mouth upto a maximum of about 1 gm. thrice daily. In hypertensive encephalopathy I.V. injection of 30-40 mgm. of the drug over a period of 2 minutes is recommended to be withdrawn at once if dizziness appears. These drugs have been used chiefly in cases of essential hypertension but have also been proved effective in renal hypertension and in toxæmia of pregnancy. The drugs cause a sudden fall in B.P., therefore one must be cautious in giving them in cases of cerebral or coronary artery disease where relative local ischæmia and possibly thrombosis may result. Again, one must note that these compounds are very potent drugs, and have caused fatal result even when given under supervision.

Quite recently veratrum viridi has come into the field as another drug for the treatment of hypertension. Its alkaloids exert a beneficial action in hypertensive cases, but how it does so is not known with certainty. A potent alkaloid of this plant is still being tried in America and elsewhere in the treatment of hypertension. Veratrum gives troublesome nausea and vomiting in the patient and is not suitable for cases of peptic ulcer. The advantages of this drug claimed over methonium compounds are that it does not cause (*i*) postural hypotension as seen in methonium compounds, (*ii*) " methonium paralytic ileus " and (*iii*) bromism.

Lastly, " neither methonium compounds nor veratrum touch the cause of the disease and both these types of preparation offer encouragement but as yet no fulfilment " (Leading article, *Lancet*, 1: 568, 1951).

Tissue Extracts—Renal extracts are still in the experimental stage. Encouraging results have been reported with hypertensinase but pyrogenic and anaphylactoid effects make evaluation difficult.

Surgical Treatment—In certain cases splanchnicectomy has been advocated for hypertension, and for this Smithwicks (1950) operation of supradiaphragmatic sympathectomy *i.e.*, removal of all sympathetic ganglion from 8D to 1L or 2L first on one side and then on the other is considered classical. Sympathectomy does not cure the disease for it cannot remove the cause, but it relieves certain symptoms like intractable headache and papillœdema and increases the expectancy of life. The criteria of its success are variable, for some consider changes in B.P. and increase in longevity and other symptomatic improvement as a success. Patients who are above 50, who have had heart failure or a stroke (confirmed by E.C.G.), severe renal damage (ascertained by high blood urea and poor renal function tests), obesity and emphysema are not to be considered for the surgical treatment, and so would not, cases of alcoholism.

The after-effects of sympathectomy are postural hypotension, faintness and giddiness on standing and temporary impotence and sterility if 2L ganglion is excised and the B.P. resumes its original level after 12-18 months of sympathectomy.

It is concluded, therefore, that operation does not effect a cure, but offers some relief of the symptoms including those of the grave ocular type.

Summary

A resume of the ætiological factors, clinical classification and therapeutic measures of hypertension is described.

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SPECIAL ARTICLE

ROLE OF AN OPHTHALMOLOGIST IN INDUSTRY

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Industry in India is expanding rapidly since the advent of independence and with it increased employment of workers has become necessary. Many of the workers must learn to handle more or less skilfully the modern fast-moving machinery, hitherto unknown to them. Since education has not kept pace with the demands of modern industry, the average worker fails to realise the necessity of safety rules laid down for his job. Thus the industrial medical officers have to face to-day problems hitherto not encountered in India. Of late, the entire problem of industrial health is being tackled with greater enthusiasm by directors of various private industries and government departments. Expenditure on more thorough voluntary medical aid to the workers have been incurred by the industrial concerns. In a recent circular the Government have stressed the importance of pre-employment medical examination.

Till recently the industrial concerns of this country have been feeling the provision of medical service a liability or an unremunerative infliction on them, and hence have been maintaining a skeleton medical staff just to attend to first-aid and left everything else to the state hospitals or to the private profession. Fortunately, the more enterprising larger concerns have realized the importance of improved medical aid, which not only looks after the first-aid in case of injuries, but also of physical examination at recruitment, and maintenance ot health of the worker and his family. When injured, except where definite hospitalisation is indicated, a thorough treatment is provided, so as to return the worker in the quickest possible time psychologically and physically rehabilitated. This, of course, necessitates maintenance of one or more fully qualified medical practitioners interested in Industrial Medicine on a full-time basis and facilities to obtain specialist opinion when necessary. Vision having been recognized as one of the most important of all human abilities and especially to an industrial worker, the services of an ophthalmologist on a part or full-time basis to large industrial concerns is very helpful.

Herein I attempt to outline the role of an ophthalmologist in industry from my short experience in some industrial concerns of Madras. I hope it will clearly explain the importance of an ophthalmic service to industry, not only to stimulate the interest of the industrialist, but also to be of some use to an average general practitioner employed as a medical officer to a firm.

The worker is seen in three phases:—(1) as a prospective employee, (2) employee in health (3) as a sick employee.

THE PROSPECTIVE EMPLOYEE

An authority has said " Approximately 10 per cent of all industrial accidents may be attributed to inadequate mechanical or machine guards and 90 per cent due to human errors or defects." Thus it is most imperative, even at the outset, when an employee is taken in, that a thorough physical examination is completed. Corrective measures if needed must be taken before he enters the firm. A thorough physical examination must include blood examination, including blood picture and serodiagnostic tests. Part of the physical examination is vision screening procedure. The actual ocular examination consists of external examination of the eyes for any pathological or congenital defects, visual acuity for distance and near vision in each eye separately, glasses worn if necessary, muscle balance and stereopsis, colour perception and appreciation, field of vision by confrontation test. Every one of the above tests is necessary to estimate an ocular efficiency for industry as would be shown subsequently. Eighty per cent of all human motions are controlled by the eyes. Hence very efficient eyes are necessary not only for prevention of accidents, but also for efficient performance of delicate and dexterous jobs. Very often ocular examination for pre-employment physical fitness certificate ends in recording visual acuity for distance. Since that alone is not sufficient, I would suggest the practice, of requiring an eye specialist's opinion on all of the above details, should become more prevalent. For grades of vision suited to various types of industry, I follow the standards suggested by Joseph Minton in his " Book on occupa-

tional eye diseases and injuries" which I have found very helpful. I am reproducing the same here.

VISUAL STANDARDS IN INDUSTRY

Grade I vision	One eye 6/6 Other eye 6/36	Workers in this group are fit for all occupations including fine, close work.
Grade II vision	One eye 6/12 Other eye 6/36	This group is fit for most occupations in Engineering industry and clerical work. Fit to drive motor vehicles.
Grade III vision	All one-eyed people with not less than 6/12 in good eye	Fir for most occupations, the safety of the good eye must always be ensured. Coal-mining, hammering, chipping and drilling should not be carried out by one-eyed people.
Grade IV vision	One eye 6/24 Other not less than 6/36	Fit for out-door occupations, carpentry, gardening, dock labour etc.
Grade V vision	All blind and partially blind, less than 6/36 each eye.	As suggested by National Institute for the blind (Britain).

N.B.—These visual standards are based on the vision of the worker, if necessary with glasses.

Prospective employees who are able to meet this standard with glasses are required to so equip themselves with glasses and should visit the eye specialist before they may be declared fit. Till such time they are classified as temporarily unfit. In my experience in Madras, this has been insisted on for two reasons, (1) Workers who, after serving for sometime with the firm, develop symptoms of eye strain, or defective vision, are required to secure eye glasses. One third the cost is borne by the company the other two-thirds deducted, in easy instalments, from the workers' salary. Obviously, with such concessions, the worker is expected to enter service after suitable correction at his own cost. (2) Studies of eye sight in factories have revealed that persons with uncorrected visual inefficiency are more prone to accidents than workers with efficient visual performance. Eye strain causes fatigue, nervousness and lowered alertness. Very often the pre-employment vision test reveals defective eye sight to the individual for the first time in his life and the necessity to wear glasses is pointed out to him. In my experience for the past one year, among 680 pre-employment examinations made, 29 were found to be temporarily unfit for want of initial correction or suitable correction. 13 men with progressive disease of the eyes or uncorrectable damage to the fundus or media were rejected.

EMPLOYEE IN HEALTH

Regular periodic health examination of workers is carried out annually. The eye examination is included. Any early pathological condition or normal senile changes such as lens opacities, degenerative changes of conjunctiva and cornea, fundus changes, changes in refraction like latent hypermetropia becoming manifest, onset or increase in presbyopia etc., are noted on the health cards and suitable remedies suggested. The prescription must be followed if the man is to remain medically fit.

Attention to visual performance helps the individual employee quite as much as it does the management. Any method which increases the efficiency of the worker and at the same time gives him comfort at his work is a real service. It should create a better attitude towards his work.

In this connection the prescription of spectacles to presbyopic workers deserves special mention. The usual working distance of the individual for his trade is an important factor. For instance, a clerk doing office work, a foreman requiring to inspect job cards and other normal reading matter in addition to technical supervision etc., would be satisfied with spectacles prescribed for the usual 33 cms. distance. Whereas a carpenter, lino-operator and men in similar jobs require their own near point vision fixed according to their trade, which would usually be more than 33 cms. Spectacles are prescribed accordingly. Our statistics show, that out of 4,500 men on an average 160 employees require eye testing and prescription of glasses every year.

During his career the worker has to face many hazards peculiar to his trade. The ophthalmologist with the Industrial Medical Officer's help, could suggest measures to guard against them. For example, proper screening measures either with glass shields or goggles against flying foreign bodies propelled by abrasive wheels, lathes, turning machines, or by hand operation such as hammering, chiselling and chipping of slag after welding operations, should be required. The goggles should not be tinted for these purposes, but made of clear glass with no flaw, no scratches, and should be light and comfortable, preferably non-splintering Triplex glass. The management should periodically inspect all glass shields and goggles and keep them in good order. Otherwise, the worker will try to avoid using them. If enforced he feels it more of an infliction than an advantage. In some western countries, if the worker fails to wear goggles during work in certain trades, he is liable to be dismissed. It is easy to find if one was wearing goggles or not while working on an abrasive wheel when numerous extremely small foreign bodies similar to one on the eye are peppered all around the eye on the lids and eye lashes. If the glasses were worn, none enter the eyes. Where ill-fitting goggles are used foreign bodies may find their way into the eye especially by the side of the bridge of the nose.

The other industrial eye hazards frequently encountered are those in welding work, effect of fumes

produced and effect of ultra-violet rays on conjunctiva and cornea causing irritation of the eyes from mild conjunctivtis, or in severe cases superficial corneal erosions. In these, there is always a latent period during which abiotic action of the rays on the epithelial cells take place with subsequent shedding off of the same and exposure of the fifth nerve-ending causing intense reflex blepharospasm. The most efficient preventive glass for this is Crookes, which cuts off the whole of the lower reaches of the spectrum and used in the darker shades reduces the glare of the luminous light as well. The welder generally uses the shield to protest his eyes when welding. But is is usually his assistant or a worker in the neighbourhood that neglects eye protection and suffers from ocular irritation. The fumes that are produced in welding seem to be irritable to the eye. Very often the worker reports to the medical centre with such a complaint. Mild astringent sedative eye drops help to relieve the pain.

Among the chemical irritants causing injuries to the eyes—alkali and acid are common, producing alkali and weak acid burns. Lime burn is the chief alkali burn that requires careful management on account of its deep penetrating quality by softening superficial layers and percolating through them. Prognostication is difficult at first. Generally, however, the condition is not very serious as only a small quantity of slaked, old lime falls in *e.g.*, an electrician or his assistant working on a plastered ceiling. The worker or the neighbour at once removes the solid particle. The rest is removed by washing at the first-aid centre. Fresh white-wash lime is, however, dangerous on account of its fluid and chemical state. Acid burns met with in automobile engineering concerns attending our centre are, however, not very serious. Dilute acids used in accumulators are the usual cause for such accidents. Intense lacrimation and the washing of the eye generally helps to clear the condition and the patient suffers from temporary irritant-conjunctivitis.

Other rarer chemical hazards that may be met with in paints and chemical factories are: amyl nitras causing corneal necrosis; aniline dyes causing chemosis and keratoconjunctivitis; beryllium as used in fluorescent lamps—keratitis; CS_2—cataract; lead—optic neuritis, accommodation difficulties etc.; tar—corneal ulcers; methyl acetate—loss of central vision; trichlorethylene as used in paint remover—keratitis, and rarely optic atrophy.

The effect of heat rays on lens should be mentioned as a possible hazard in case of workers dealing with intense heat *e.g.*, furnaces, though such instances are not common and take a long time to develop. Heat absorbed by the cornea and aqueous humour are quickly lost and no effect on those are perceptible, whereas the lens absorbs heat and it is unable to lose it quickly with the result that changes take place causing the appearance of cataract, *e.g.*, chain makers' cataract and glass blowers' cataract etc.

Another important detail of prevention of general and ocular accidents is in connection with illumination. The right amount and type of illumination for a particular job is essential for comfort and efficiency. To assure visual comfort, the indirect system of lighting is the safest. Comfort can be obtained in relatively dim light if uniformity of brightness is achieved. The indirect system is best suited for offices. The contrast between the upper and lower parts of the room should be reduced by using light colours on furnitures and floor. Direct system requires expert care. Naked bulb showing out in contrast to ceiling should be avoided. With regard to the colour in light, a yellowish tint is preferable (the colour of highest visibility in spectrum to light adapted eye). The colour of the artificial light used in deep rooms, where it mixes with natural light to increase the illumination of the place, should be such that the two should be more or less similar, and the natural choice is a fluorescent lamp. One disadvantage with fluorescent lamp operating on A.C. current is the cyclic variation of light which can give rise to stroboscopic effect with moving objects which in bad cases may appear to alter the speed or even seem to stop a rotating wheel. By suitable electrical circuit arrangement this effect can be minimised. There is no evidence that light fluctuations of lamps on the ordinary circuit have a deleterious effect on the eye. But glare due to light from an unwanted angle is very annoying to the worker, reducing his efficiency and hastening the onset of fatigue. Hence direct lights should be properly screened to avoid glare.

With regard to colour on walls etc., soft greyish hues are best. They lack aggression and do not distract. Where temperature is high, cool colours like green and blue could be used, and cream colour where temperature is low. A soft blue green fulfil most requirements.

Sick Employee

The sick employee can be classified into those suffering from (*a*) accidental injuries and (*b*) other diseases of the eyes.

(A) Accidental Injuries:—In spite of all precautions taken to prevent, accidents are inevitable. All efforts should be directed towards reduction of their number and severity. From the statistics for the year 1950-51 of injuries from industrial concerns under study 476 sustained ocular injuries which works out to 30 per cent of all injuries reported. The industrial concerns on an aggregate employ about 4,500 workers. Fortunately none of the injuries led to loss of vision sufficiently serious to impair working efficiency.

The chief cause of eye injuries in order of frequency have been due to (1) flying objects *viz.*, (*a*) those set in motion by hand tools (*b*) those set in motion by abrasive wheels giving rise to small flying particles, with more or less equal frequency: (2) irritation of eyes from exposure to fumes *e.g.*, welding; (3) dust and grit *e.g.*, while servicing the under chassis of motor vehicles

etc.; (4) irritant and corrosive substances like brake fluid, dilute acids from accumulators, lime, paint solvents etc.; (5) irritant conjunctivitis or very rarely superficial corneal erosions from exposure to ultraviolet rays emitted during welding operations—usually the assistant of the welder suffers. Workers wear goggles while working on abrasive wheels and in turning operations, otherwise flying particles from other kinds of work, for example hammering, chipping off slag, scraping of old paint etc., are not usually screened off by any protective means. Even where arrangement for protection of the eyes exist such as glass shields, or wearing of goggles, some workers either neglect to make use of them or fail to understand their proper use. Instances have come to our notice where the worker looks over the shield to get close to his job in his anxiety to do his best, thus exposing himself to injury by foreign bodies.

These injuries could be classified as *major* and *minor* eye injuries. Fortunately most of the injuries are of a minor nature and major ones are extremely rare. Minor ocular injuries are an every day occurrence even in places where protective measures are adequate and strictly adhered to.

Majority of injuries are to the cornea with foreign body 48·2 per cent of all ocular injuries reporting at the medical centre with either only an abrasion of cornea giving rise to a foreign body feeling, or an actual foreign body embedded to various depths in the corneal tissue. Most of them are metallic foreign bodies. Of lesser frequency are foreign bodies lodged in the conjunctiva, usually in the upper sulcus subtarsalis or at the inner canthus. Fortunately, the practice of getting first aid from the neighbour has become rare and most of the workmen report as soon as ocular injury occurs to their immediate superior who directs them to the medical centre for treatment. This fact and immediate use of antibiotics have been responsible for the quick return of the worker back to industry with little or no loss of efficiency. Since treatment for corneal abrasion and foreign bodies entails bandaging the eye the worker is taken off duty for the rest of the day. The vast majority of the patients are ready for work, the next day. Very little scarring is left on the cornea and as the injury is rarely over the pupillary area, there is no damage to vision in most of the cases. In case of foreign body with rust ring, if the latter is not thoroughly scraped out on the first day, the irritation and lacrimation still continue on the second day when the patient feels as if the foreign body still existed. In such cases the rust ring would have to be removed and period of absence extended accordingly.

One group of firms have accepted the principle of paying the workers full wages and dearness allowance during a period not exceeding six days of certified absence after an industrial accident. Thereafter Insurance pays only wages. Such a gesture in the long run helps the company in that the worker gets a thorough treatment without having to hurry back for fear of losing wages and thus gets back to work fully rehabilitated both physically and psychologically.

Special mention however must be made about management and ocular affections of welders. Welding fumes consist of oxides of nitrogen, CO_2, CO, SO_2, fluorides, phosphin, arsine etc., and where the job has to be done in confined spaces as for instance on the inside of a tank etc., the fumes are not readily dispelled and the same may irritate the eyes and in serious conditions lead to what is known as metal fume fever. The irritant effect on the eyes, easily pass off without any treatment. Mild astringent-sedative drops may, however, be prescribed as a placebo. Acute arc eye is a rare condition. In more than a year's experience as an ophthalmologist to a group of engineering concerns employing over 4,500 workers and where welding (gas and electric) is carried on day in and day out I have come across only a single instance of genuine acute arc eye, with superficial corneal erosion appearing after a latent period after welding. Such a case is best treated with frequent irrigation with cold saline and guttæ adrenaline 1 in 1,000 instillations and if the patient complains of great deal of pain pantocaine 1 per cent should be instilled. Antistine with vasoconstrictors (Antistine-privine, Ciba) is beneficial. The patient has to be put off duty for the rest of the day and, if necessary, a little longer.

Major eye injuries that might happen in industry include those where there is rupture of the coats of the eyeball or serious intraocular damage on account of injury with blunt or sharp objects with consequent impairment of vision, or extra-ocular damage to any extrinsic ocular muscle with consequent diplopia, temporary or permanent. Such injuries are, however, rare on account of the usual precautions taken by the management and the worker. When such injuries take place the patient should be put under expert care as soon as possible and appropriate surgical treatment adopted to minimise the effects of damage. It is in connection with effects of major eye injuries that the problem of compensation usually arises. *Appraisal of visual efficiency and computation of compensation—Total permanent disability of both eyes≡Total permanent disability of the individual.* To estimate full visual efficiency three co-ordinate individual factors should be taken into account. They are: (*a*) visual acuity. (*b*) field of vision, (*c*) muscle function.

(*a*) The best visual acuity with correcting glasses should be noted (6/6=100 per cent: 6/60=20 per cent; 1/60=1 per cent). If more than 4 D anisometropia exists between the injured eye and the fellow the either best possible vision of the injured eye without corrections or with lenses of not more than 4 D Sphs. difference from the fellow eye shall be acuity that is taken into account.

(*b*) Field of vision with white target subtending 1° under illumination of not less than 3 f.c.—the amount of radial contraction in 8 principal meridians

is determined, the sum of these divided by 420 gives visual field efficiency of one eye in per cent.

(*c*) Muscle function. An industrial diplopia chart is used which is divided into 20 squares—the number of squares in which diplopia occurs is found and if there is diplopia in all squares 50 per cent efficiency and so on.

The industrial visual efficiency of one eye is obtained by multiplying the per cent efficiency thus found of the three co-ordinate factors. Example—If VA = 40 per cent, field = 81 per cent and motor field efficiency = 100 per cent, then $0{\cdot}4 \times 0{\cdot}81 \times 1{\cdot}0 = 32{\cdot}4$ per cent is the industrial visual efficiency of the eye.

Industrial visual efficiency of the individual could then be calculated as follows:

Industrial visual efficiency of the less efficient eye *plus* 3 × industrial visual efficiency of the better eye, *divided by* 4. Example—If the injured eye efficiency is 40 per cent and better eye 100 per cent then $\frac{40 \textit{ plus } 300}{4}$ = 85 per cent is the industrial efficiency of the individual and the compensation would be 15 per cent of the amount due to him if he suffered total permanent disability.

In our state, according to Compensation Act, loss of one eye or complete and permanent loss of use of one eye, is said to cause 30 per cent loss of earning capacity of an individual and compensation due to the worker is calculated thereon.

(B) Other Diseases of the Eyes: A sick employee suffering from other diseases of the eyes requires the same amount of attention and care as when he is involved in an industrial accident. If his condition is infective he is given adequate leave and asked to attend for treatment daily. If the sources could be traced in his family he is encouraged to bring them as well for treatment. Provision for treatment of family of the worker is helpful in two ways (1) that it prevents spread of infection to the worker if he is not already suffering from the condition; (2) it saves, worry, money, and time of the worker which would otherwise be spent in making arrangement for their treatment, thus indirectly benefiting the worker and management.

In non-infective cases adequate treatment is carried out, if possible, as an out-patient, otherwise he is directed to a nearby hospital.

In this brief resume of the role of an opthalmologist in industry, I have hoped to focus the attention of the industrialist and industrial medical officer on the importance of ocular care in industry. Where possible an ophthalmologist's service, either part time or whole time, should be arranged. His services should be very valuable: (1) In ocular examination of a prospective employee and making full and correct notes about his condition in giving an opinion as to his ocular fitness or otherwise; (2) In suggesting measures for the preventive aspect of accidents, proper illumination etc.; (3) In advising treatment of all or at least difficult ocular accidents; (4) For opinion as regards ocular compensation claims; (5) And in attending to the needs of a sick employee and his family, including refraction work.

APPENDIX A

Hints for the First Aid Treatment of External Ocular Injuries in Industry

(To be carried out only in the Medical Centre).

1. *Corneal Abrasion.*

(*a*) Instil 2 drops of fluorescein solution into the eye, and then give a small gentle wash. Abrasion stains green.

(*b*) Press over the sac region and see if any regurgitation occurs through puncta. If doubtful, test for patency of lacrimal passage.

(*c*) If passage free—two drops of albucid 10 per cent or steramide 10 per cent solution followed by penicillin eye ointment 1,000 units per gram and then apply pad and bandage.

(*d*) If passage is not free—apply albucid drops and ung. penicillin. Do not bandage and *case to be seen by the eye specialist.*

2. *Foreign Body in the External Eye.*

(*a*) Patient lying on a couch, inspect cornea with oblique illumination and loupe. (Fluorescein instillation helps). If cornea is free evert the upper lid and look for foreign body there, if found remove with a pledget of moist cotton wool. If not there give a thorough saline wash to the eye and any loose foreign body anywhere else comes out.

(*b*) If on cornea—anæsthetise with pantocaine 2 per cent solution. Under aseptic conditions remove foreign body with sharp needle. If rust ring is present try to scrape it out. If it does not come off leave it for the eye specialist. Then test for regurgitation by pressure over sac area, if no regurgitation, instil albucid 10 per cent drops. Ung. penicillin or steramide, pad and bandage. *Latter to be kept for 24 hours.*

(*c*) Difficult rust ring and deeply embedded foreign body to be left alone for the specialist to see after applying albucid drops, ung. penicillin and pad and bandage.

3. *Burns of Conjunctiva and Cornea* (Splashing of hot molten metal into eyes in foundries).

Immediate application of albucid drops and ung. penicillin in sufficient quantity to fill the conjunctival sac and then pad and bandage. If there is any solid particle on the cornea that of course should be removed first.

4. *Contusions of the Eye* (Large bodies hitting the eye).

In Minor Injuries—" Black eye," corneal abrasion, subconjunctival hæmorrhage.

Severe cases—Rupture of the globe—intra and retro-ocular lesions etc. If possible, note down the vision of the injured eye. Albucid drops, ung. penicillin apply, pad and bandage. *To be shown to the eye specialist.*

5. *Chemical Eye Injuries.*

(*A*) Chemical Splashes	1. Ammonia, NaOH, other alkalies. 2. Ammonium sulphate and other fertilizers. 3. Nitric acid. 4. Sulphuric acid and other acids. 5. Lime and cement splashes.

(*B*) Chemical gases and vapours ...	1.	Ammonia.
	2.	HS M_2S.
	3.	Chlorine.

Acid solutions: (Severe burns of cornea and conjunctiva immediate damage. Does not increase in depth in due course).

(*a*) Immediate saline wash. To be prolonged and thorough (minimum 10 minutes wash).

(*b*) If cornea is damaged. Atropine, ung. penicillin in plenty. Pad and bandage. *To be shown to eye specialist.*

Alkalis: (Most dangerous. Slow action increases in depth. Prognosis to be guarded and reserved till end).

(*a*) Saline or better still with 5 per cent ammonium chloride solution. Wash to the eye. Thorough and prolonged—minimum 10 minutes.

(*b*) Remove any solid particles of chemical or loose epithelium. Repeat wash with saline or ammonium chloride.

(*c*) Albucid drops and ung. penicillin, pad and bandage. *To be shown to eye specialist.*

Acute "Arc Eye."

1. Instillation of solution adrenaline 1: 2,000 and frequent irrigation with cold saline solution.
2. Cold eye compresses.
3. If there is great deal of pain—pantocaine drops 1 per cent.
4. Antistine-privine (Ciba) drops.
5. Further instillation Arc eye lotion.

Zinc sulph ...	...	... gr. i
Boric acid ...	...	... gr. iv
Sol. Adrenaline Hydrochlor (1 in 1,000)		oz. 1.

6. Dark glasses.

APPENDIX B

EYE ACCIDENTS—CLASSIFIED BY TRADES
FROM APRIL 1950—APRIL 1951

Trade	*No. of Accidents Reported*	*Leave Granted in days*
Blacksmith ...	12	18 −2½ H.
Hammerman ..	9	13½
Mason	1	½
Mechanic	10	20
Tinker	21	31
Painter	5	10
Turner	24	42
Hydraulic press ..	4	4½
Gas fitter	3	4½
Blower boy ...	2	1
Carriage fitter ..	1	4
Electrician ..	1	5½
Welder	12	24
Carpenter ..	6	11½
Driller	4	4½
Sawyer	5	6½
Bench fitter ...	6	6
Supervisor ...	1	3
Engraver ...	1	2
Machineman ...	1	3½
Reborer	1	1
Liner	1	1
Greaser	1	½
Labourer	2	3
TOTAL ..	134	221½ −2½ hours

CASE NOTES

ANAPHYLACTOID SYMPTOMS IN A BABY DUE TO SUCKING OF MILK FROM THE MOTHER IN ANAPHYLACTIC SHOCK

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A female, aged 25 developed anæmia at the 9th month of her third pregnancy. Her Hb. was 45 per cent (Hellige) and colour index 1·1. She was treated with iron by mouth and injections of liver extract and vitamin B_{12}. Two c.c. of anahæmin was injected I.M. on 28-7-51, 3-8-51 and 9-8-51 and one c.c. of Combex parenteral was injected on 12-8-51 and 15-8-51. She gave birth to a baby on 17-8-51 and her Hb. then rose to 55 per cent.

On 7-9-51 for further improvement of her blood picture, 1 c.c. of anahæmin was injected I.M. at 3-30 P.M. Within 5 minutes of the injection, she complained of intense itching all over the body and she started scratching. General flushing and urticarial rashes appeared all over the body almost immediately. There were cough and breathlessness and she complained of a choking sensation in the throat. This was accompanied by severe prostration. Pulse was 130 per minute with small volume and low tension. Respiration was 28 per minute and an expiratory wheeze was audible even without the stethoscope.

½ c.c. of adrenaline injected immediately was followed by some improvement. The injection was repeated within 45 minutes. Calcium gluconate powder 1 drachm was also given by mouth. Anti-histaminic drugs were not readily available and could not be given. The condition of the patient improved considerably within half an hour of the 2nd injection. Her temperature was found to have risen to 101°F in the evening. Gardinal 1½ grain was given at bed time to ensure better sleep. Next morning she was much better but she complained of severe weakness which passed off gradually in course of the next 36 hours. A blood slide examined during convalescence revealed an eosinophilic count of 14 per cent. Her eosinophil count during and before delivery was only 1 per cent. No past history of allergy in herself or in her family.

Baby 21 days old and suckling mother's milk as the only food became very irritable and fretful the same evening (when its mother got the reaction). It slept badly during the night. Next morning its axillary temperature was found to have risen to 100°F. A scarlatiniform rash was noticed all over the body particularly on the chest and abdomen. An expiratory wheeze was detected on examination of chest. The blood eosinophil count was 4 per cent. These menifestations gradually passed off in the course of one day.

DISCUSSION

Two aspects of the case deserve consideration:

(1) That during an anaphylactic shock, the substance which is responsible for development of shock is excreted with milk.

(2) That a new-born baby on taking such milk may develop symptoms of anaphylaxis.

It is now agreed by most that anaphylaxis is due to the combination of antigen with antibody inside cells which damages the cells and may lead to the release of histamine and other toxic substances. The pharmacological effects of histamine are dramatic even in small doses and they may be summarized as contraction of most plain muscles, dilatation of capillaries and secretion of most glands, particularly the acid-secreting glands of the stomach.

The authors do not know if any work has been done to see if histamine is excreted with the milk during an anaphylactic shock of the lactating mother. The concentration of histamine in the blood has been estimated pharmacologically but the results have been mostly disappointing, chiefly because blood is such a complicated fluid. The estimation of histamine in the urine has been the most sensitive way of detecting the liberation of histamine in the body. The urine normally contains free histamine and the inactive substance acetyl-histamine. After intravenous injection of histamine or during anaphylactic attacks, the amount of free histamine in the urine is increased. It remains to be seen if free histamine is excreted with milk during anaphylactic reactions.

The other point of discussion in the case is the occurrence of symptoms in the baby after ingestion of the milk. Now histamine when taken by mouth usually produces little pharmacological effects. The mucous membrane of the gut contains particularly large amounts of the enzyme histaminase (which destroys histamine) and this probably plays an important part in protecting the body from the effects of absorption of histamine. Histamine taken by mouth is also partly conjugated in the intestine or liver and increased amounts of conjugated histamine appear in the urine.

Larger amounts of histamine, however, taken by mouth may produce the effects of histamine. It has been shown that when histamine is given by mouth there is a small but definite increase in the amount of free histamine in the urine. There is also a possibility that new-born babies are too much sensitive to ingestion of histamine or that in new-born babies the function of destruction of histamine or its conjugation may not be so well functioning as in adults.

SUMMARY

(1) Symptoms of anaphylaxis occurred in a baby due to sucking of milk from the mother then suffering from an anaphylactic shock. This is obviously possible if histamine is excreted with the mother's milk.

(2) Symptoms occurring in the baby seem obviously to be due to ingestion of histamine with the milk. The possibility is raised if new-born babies are too much sensitive to ingestion of histamine or the function of destruction or inactivation of histamine when taken by mouth is not so well developed in new-born babies as in adults.

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TOXICITY OF THIAMINE HYDROCHLOR

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Very recently I came across a case of toxic reactions following injections of thiamine hydrochloride. Different foreign brands were used but with the same untoward effect.

The patient, a Hindu bachelor, aged about 30, the youngest son of a very large family, gave the following history of his present illness. He had a peculiar painful sensation in his left arm. There were anæsthetic patches and paresis on the lateral part of his hand. The muscles of the supraspinous region showed wasting. The ailment started, about 7-8 years back, just following a long bout of hard cycling on a chilly night. He was advised by his doctors to apply a liniment in the arm and in the shoulder, and to have a course of vitamin B_1 injections along with injection of polydin. He had an uneventful recovery. Unfortunately, after 2 years the patient had a relapse; this time, he was treated by some local applications and a course of thiamine hydrochloride (benerva) and glucoldin. He required a little longer treatment on this occasion; all the same, he became clinically allright. After another two years the patient had another relapse; possibly all these relapses were due to some septic focus, may be the septic gum, which was the original source of the mischief, and as that was not attended, no lasting benefit was obtained. However, the strangeness of the case was noticed, when he started treatment for his second relapse. The first injection of vitamin B_1 gave him a peculiar toxic symptom with ultimate collapse and sinking of the pulse. For the next injection, he changed both his doctor and the brand, but unfortunately the reaction this time was worse and the doctor refused to give him any further vitamin B_1 injection. An interesting point to be noted in this connection is that the contents of the same vial of vitamin B_1 administered to other patients produced no toxic reaction.

The picture that the patient exhibited during the time of a reaction, is as follows—His face, the bald of his head, his neck and all other exposed portions of his body became intensely suffused with blood and looked flushed. His eyes became red and showed a tendency to lacrimation. The nose became stuffy and the stuffiness continued even after the reaction passed off. The pulse rate went up, it became weak and quick, the B.P. appeared to sink down. The patient appeared to be stunned and sometimes after the passing away of the reaction, the patient remained mum and confused for a few hours more, though during the reaction he seldom lost his consciousness and was able to give monosyllabic answers to any question asked.

The patient's subjective feeling was that of complete collapse and sinking. He felt very much restless within and had an unbearable urge for evacuation of the bowels and the bladder. The most important thing to note is that as soon as the reaction passed off, the urge for stool and urine disappeared automatically. During the reaction phase, the patient felt a peculiar distressing warmth all over his body. An itchiness started and it went on in a fleeting fashion in all the different parts of the body. The condition lingered for about 15 to 20 minutes even after administration of antistine and ephedrine hydrochloride.

POINTS OF INTEREST

A few interesting points to be noted in connection with the case are—

1. All along, the patient received 100 mgm. of vitamin B_1 I.M. and the series always ended with 10 to 15 injections at the utmost.

2. On the first two occasions he had no reaction.

3. On the third and subsequent courses, separated by months and years, he showed reactions of a very marked type, irrespective of the brand used. Further, the same drug never gave any toxic symptoms to control cases.

4. The reaction was not simply like the ordinary book picture of vitamin B_1 toxicity such as tachycardia, weakness, tremor etc. The picture was that of complete collapse with signs and symptoms suggestive of nicotinic acid hypersensitivity. Contrary to what the books suggest *viz.*, that continuous use of the drug is responsible for the toxic effect, the patient showed the reaction on the very start of the courses. If the reactions are to be considered as simply allergic why then had he not such allergic reactions during the first two courses of treatment, though it is to be noticed that the patient showed almost no reaction or very mild reaction, if just before the injection, he swallowed one tablet of antistine and one of ephedrine.

5. Even in the third or subsequent courses after two injections, he did not show much reaction, in the remaining portions of the series, but whenever a fresh course was started all his reactions came back in full swing.

The observations recorded are very interesting. It is generally believed that vitamin B_1 is harmless and does not ordinarily give rise to any unpleasant symptoms even when used for a prolonged period. It has, however, been noticed to produce flushing of the face and an uncomfortable sensation, some brands more than others. In some cases full therapeutic doses (40 to 50 mgm. daily) may give rise to symptoms of hyperthyroidism, *viz.*, tachycardia, tremor and nervous irritability. Laws, C. L. (*J.A.M.A.*, **117**: 176, 1941) and Schiff L. (*J.A.M.A.*, **117**: 609, 1941) reported sensitization after repeated intravenous injection with symptoms similar to anaphylaxis with collapse. Reingold, I. M. and Webb, F. R. (*J.A.M.A.*, **130**: 491, 1946) recorded sudden death with symptoms of collapse following intravenous injection of 100 mgm. Large doses of any of the individual members of the vitamin B complex alone, specially of thiamine, may lead to other vitamin B deficiencies. An increase of one member of the complex should be accompanied by an increase in all other factors.—ED., *J. Indian M. A.*

Dr. Bauer, President, A.M.A. & Secretary General, W.M.A., to visit India

Dr. L. H. Bauer, President of the American Medical Association and Secretary General of the World Medical Association will be coming to India for a short period and visit Delhi. Bombay, Madras and Calcutta.

Dr. Bauer's programme will be as follows:

Wednesday, 22nd October, 1952—Arrival in Delhi.

Thursday, 23rd October, Friday, 24th October—Address members of the Working Committee, members of the Profession and general public, hold Press Conference, meet Ministers etc. Definite timings to be fixed by the Central Office, I. M. A. and the Delhi Medical Association.

Saturday, 25th October, Sunday, 26th October—Leave for Bombay at 8-30 A.M. and reach Bombay at 12-30 P.M. Halt at Bombay. Address members of the profession and meet Ministers etc. Definite bookings and arrangements to be made by the Bombay Branch and Bombay Provincial Branch, I. M. A.

Monday, 27th October, Tuesday, 28th October—Leave for Madras at 7-10 a.m. and reach Madras at 11-5 a.m. Halt at Madras. Address members of the profession and general public and hold Press Conference, meet Ministers etc. Definite bookings and arrangements to be made by the Madras Branch and South Indian Provincial Branch, I.M.A.

Wednesday, 29th October, Thursday, 30th October—Leave for Calcutta at 8-30 a.m. and reach Calcutta at 1 p.m. Halt at Calcutta. Address Members of the Profession and general public and hold Press Conference, meet Ministers etc. Definite bookings and arrangements to be made by the Calcutta Branch and Bengal Provincial Branch, I.M.A.

Friday, 31st October—Departure.

Dr. Bauer will speak in particular on the medical care plans in U.S A. and the part played by the American Medical Association in rendering adequate medical aid to the people.

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INDIAN MEDICAL ASSOCIATION
CALCUTTA, OCTOBER, 1952

PREMATURE BIRTH AND RETROLENTAL FIBROPLASIA

For some years past attention has been drawn to a peculiar condition affecting the eyes of infants born prematurely before full-time pregnancy. The close relation between this abnormal eye condition and prematurity was first pointed out in 1942 by Terry[1] who referred to this condition as "fibroblastic overgrowth of persistent tunica vasculosa lentis," and later on as "retrolental fibroplasia." Dunham[2] has remarked that, for several years previous to Terry's reports, sporadic reports had been recorded about cases with similar pathology in full-term infants, children and even in adults. Numerous cases have been reported, and several studies undertaken, in recent years, specially in U.S.A., about this peculiar eye condition of retrolental fibroplasia of prematurity.

The disease usually makes its appearance between three to six months of neonatal life of a premature infant, the mother perhaps notices some abnormality about the eyes—white spots in the eyes, or the infant fails to recognise its mother or follow objects moved before its eyes. On further examination, there may be nystagmus, microphthalmus, photophobia, strabismus, impaired vision and fœtal blue colour of the iris. Ophthalmoscopic examination often shows a shallow anterior chamber, a greyish white opaque membrane behind the lens with thin ciliary processes in front of the opaque membrane. There may also be found a persistent hyaloid artery, retinal hæmorrhages with separation of the retina. The condition is usually bilateral. Owens and Owens[3] undertook a systematic ophthalmoscopic study of 111 premature infants none of whom showed retrolental fibroplasia at birth. They have described the stages of development of retrolental fibroplasia; starting with slight gradual dilatation of the retinal vessels, the veins more than the arteries; later these, specially the arteries, become tortuous. These are followed by small peripheral greyish white elevations of the retina, with slight basal pigmentation and tortuous retinal vessels coursing over them. The disc margins become blurred with generalised retinal œdema. Many more such greyish areas quickly develop scattered over the fundus and a grey membrane proceeds centrally in folds from the periphery of the retrolental space. Numerous vessels are seen over this membrane which seems to be the anterior portion of the swollen retina, from the detached portions of which bands of tissue extend into the vitreous. By gradual extension and fusion of these peripheral folds of the retina, a complete retrolental membrane is ultimately formed, with broad ciliary processes extending on to it at its periphery and numerous vessels, appearing to radiate from the centre, on the surface of the membrane. There are occasional small hæmorrhages from these vessels. The anterior chamber becomes shallow, with formation of posterior synechia and a greyish atrophic appearance of the iris. In their series the first abnormality was detected at 80 days of life, a complete membrane developed in one case in 105 days, and in no case did a membrane develop after 5½ months.

Kinsey and Zacharias[4] made a retrospective study of infants weighing under 4 pounds or less at birth in two different cities with 22 per cent and 7 per cent incidence of retrolental fibroplasia; and the incidence was still higher (26 per cent and 23 per cent) amongst the group with lesser birthweight of 3 pounds or less. Paul[5] found an incidence of 4·4 per cent in a series of 223 infants weighing less than 2,500 gm. Crosse[6] found an incidence of 0·8 per cent in infants under 4 pounds and 4·2 per cent amongst those under 3 pounds in a follow-up study of 4,000 premature births at Birmingham. Owens and Owens[7] found that retrolental fibroplasia occurs in about 15 per cent of the infants with birthweights of 3 pounds or less. Laupus, Bousquet and McLean[8] found an incidence of 14 per cent of retrolental fibroplasia before six months of age amongst 57 infants weighing less than 1,650 gm. (about 3½ pounds) at birth. Gilger[9] found 3 per cent incidence of retrolental fibroplasia among 229 infants weighing 5 pounds or less at birth with an incidence of 7·3 per cent in 96 infants weighing less than 4 pounds. Exline and Harrington[10] found no case of retrolental fibroplasia on examining, after seven months, a series of 80 infants, (83 per cent negro people) weighing less than 1,500 gm. at birth. Reese[11] estimated that one-third of all cases of blindness in pre-school children were due to retrolental fibroplasia and Kerby[12] found it responsible for 1·5 per cent of the cases of blindness in school children, based on records of 3,905 eye-examinations. Moffat[13] reports an incidence of 12 cases of retrolental fibroplasia in a follow-up study of 119 children in institutions for the blind; all the 12 cases weighed less than 3 pounds 8 ounces at birth. Hipsley[14] reports an incidence of 5 cases in a study of 97 surviving infants with birth-weights of 3 pounds or less. Brown and Corner[15] report 16 cases in 6 years among 434 surviving babies weighing 4 pounds 6 ounces or less at birth.

[1] *Am. J. Ophth.*, **25**: 203, 1942.
[2] *Bull. World Health Organ.*, **4**: 605, 1951.
[3] *Am. J. Ophth.*, **32**: 1, 1949.
[4] *J. A. M. A.*, **139**: 572, 1949.
[5] *Arch. Ophth.*, **41**: 659, 1949.
[6] *Proc. Roy. Soc. Med.*, **43**: 232, 1950.
[7] *Am. J. Pub. Health*, **40**: 405, 1950.
[8] *Am. J. Dis. Child.*, **81**: 617, 1951.
[9] *Am. J. Ophth.*, **32**: 917, 1949.
[10] *J. Pediat.*, **38**: 1, 1951. [11] *Arch. Ophth.*, **41**: 527, 1949.
[12] *Sight-Saving Rev.*, **20**: 67, 1950.
[13] *Proc. Roy. Soc. Med.*, **43**: 223, 1950.
[14] *M. J. Australia*, **1**: 473, 1952.
[15] *Brit. J. Ophth.*, **36**: 281, 1952.

Reese[11] examined 3 eyes and found fibrous tissue, rich in vascular channels, around the base of the vitreous; mostly due to hæmorrhage and organisation with subsequent contraction causing detachment of the retina. Klien[16] examined 3 eyes and found remnants of the primary vitreous containing angiomatous blood vessels, with retinal detachment in one eye. Heath[17] examined ocular tissues removed at autopsy and concludes that the retrolental fibroplasia occurring in premature infants is a secondary manifestation of primary retinal disease which is an œdematous, hæmorrhagic and proliferative process with accumulation of vascular tissue in the retina. Baar[18] has reported detailed histological findings similar to the above, but with no synechia nor any inflammatory changes in the iris. Wolff[19, 20, 21] stresses that the retrolental membrane is inflammatory. He believes that a "noxious stimulus" reaches the embryo very early, probably in the first few months of intra-uterine life; its effect on the eye is primarily on the retina, which remains non-attached in part or as a whole. Bembridge and Jackson[22] described nine cases studied pathologically, and their findings conform with those of Wolff. Rubinstein[23] studied three enucleated eyes with histological findings similar to the other observers.

Retrolental fibroplasia has to be differentiated from retinoblastoma (glioma of the retina), pseudoglioma, congenital cataract, persistent hyaloid vessels, persistent hyperplasia of the primary vitreous, congenital toxoplasmosis and dysplasia of the retina (Krause[24]). The association with prematurity, absence of ocular findings at birth, gradual onset after about three months and formation of the membrane by about six months, the negative tests for toxoplasmosis and affection of both eyes, usually serve to settle the diagnosis. In this connection, Bembridge *et al*[25] have drawn attention to certain differences in ophthalmoscopic appearances between the infantile and the adult eye. "The infantile optic disc is always extra-ordinarily pale and the central fundus often has a patchy distribution of pigment. The retinal periphery constantly has a peculiar œdematous-looking greyness, gradually merging into the pinkness of the posterior fundus." These appearances are seen in both full term and premature babies, and disappear in a few weeks after birth.

The ætiology of retrolental fibroplasia still remains obscure. No common causative factor has been found which can account for all, or even the majority, of these cases. Numerous prenatal causes affecting the mother have been thought to contribute to the disease, such as intrauterine bleeding, maternal age and parity, multiple pregnancies (twins etc.), race, sex of the infant, Rh factor, chronic illness of the mother, maternal virus infection (rubella) during pregnancy, toxoplasmosis and vitamin A deficiency of the mother. On the other hand, a large number of other factors operating postnatally have also been incriminated. Of these, mention should be made of deprivation of maternal hormones because of premature birth, precocious functioning of systems such as respiratory, digestive, cardiovascular and heat regulatory; precocious exposure to light; inability to assimilate vitamins, birth-trauma, physiological anæmia of prematurity and intra-ocular inflammation. Reese[11], Klien[16], Speert *et al*[26], Kinsey and Zacharias[4], Gregg[27] and several others have shown evidences suggesting various prenatal factors as being responsible for the causation of retrolental fibroplasia of prematurity. Hepner and his coworkers[28] found that certain manifestations of the disease were congenital in origin, while others had a postnatal origin. They found no influence of routine sulfonamides or of a change in iron-dosage on the incidence of retrolental fibroplasia. They also found that the use of a multiple vitamin preparation containing a synthetic emulsifying agent appeared to raise the incidence of the disease. Gilger[9] found no correlation between the incidence of the disease and maternal age, parity, frequency of uterine bleeding, virus infections, chronic illnesses during pregnancy, race or cause of premature labour. Terry[29] regarded precocious exposure to light as the most important factor. Owens and Owens[3, 30] consider postnatal influences, specially deficiency of vitamin E as responsible factors. However, Bembride *et al*[25], Reese and Blodi[31] and Kinsey and Chisholm[32] could not substantiate the charges against vitamin E deficiency. Withdrawing of vitamin A and iron (which inhibit the availability of vitamin E) and giving of vitamins D and C in water-miscible form had also no effect on the incidence. Kinsey and Zacharias[4] found a striking rise in the incidence of the disease according to the increase in the number of days the premature baby was kept in oxygen. Campbell[33] in Australia also found evidences suggestive of oxygen toxicity, with a significantly higher number of cases in a hospital where oxygen was more freely used than in another. According to Szewczyk[34] the whole picture can be explained by anoxia. He suggests that a relative anoxia is possibly produced after acclimatising the infant to high oxygen tensions. This hypothesis of an ischæmic mechanism is corroborated by the findings of Rubinstein[23] and also supported by Crosse[35]. Reese

[16] *Arch. Ophth.*, **41**: 553, 1949. [17] *Ibid*, **44**: 245, 1950.
[18] *Proc. Roy. Soc. Med.*, **43**: 233, 1950. [19] *Ibid*, **43**: 227, 1950.
[20] *Am. J. Ophth.*, **33**: 1768, 1950.
[21] *Brit. M. J.*, **1**: 924, 1952. [22] *Ibid*, **2**: 1484, 1951.
[23] *Brit. J. Ophth.*, **36**: 303, 1952.
[24] *Arch. Ophth.*, **36**: 387, 1946.
[25] *Brit. M. J.*, **1**: 675, 1952.

[24] *Am. J. Ophth.*, **34**: 1649, 1951.
[25] *Ulster M. J.*, **21**: 32, 1952.
[26] *Am. J. Obstet. & Gynec.*, **59**: 246, 1950.
[27] *Tr. Ophth. Soc. Australia.*, **3**: 35, 1941.
[28] *Pediatrics*, **5**: 771, 1950.
[29] *J. Pediat*, **29**: 770, 1946.
[30] *Am. J. Ophth.*, **32**: 1631, 1949. [31] *Ibid*, **34**: 1, 1951.
[32] *Ibid*, **34**: 1259, 1951. [33] *M. J. Australia*, **2**: 48, 1951.
[34] *Am. J. Ophth.*, **34**: 1649, 1951.
[35] *Ulster M. J.*, **21**: 32, 1952.

and Blodi[21] have suggested deficiency of adrenocortical hormone as a possible cause. Further studies only will determine the causative factors. At any rate, the increased incidence of retrolental fibroplasia cannot be completely explained by the recent improvement in survival rate among infants of low birth-weight.

It is easily appreciated that while the ætiology of retrolental fibroplasia is still obscure and the pathology not clearly understood, attempts at prevention and therapy can only lead to partial success. Variations in administration of vitamin A, covering the infants' eyes with cotton pads (Hepner *et al*[24]) and administration of vitamin E have all been tried and failed to prevent the incidence of retrolental fibroplasia. Treatment of the advanced stage of this disease has been attempted surgically, with diathermy, by roentgen irradiation and also with drugs—in an attempt to prevent complications like glaucoma and posterior synechia. Szewczyk[34] has suggested that infants who show early signs of retrolental fibroplasia might be cured by returning them to a high concentration of oxygen. Reese and Blodi[21] treated a series of fourteen early cases with ACTH by using a dosage between 20 to 25 mgm. daily for 14 days. Later, Blodi *et al*[37] reported the results of treatment of 19 infants with ACTH for periods of 9 to 25 days. ACTH has been used for this condition subsequently by many other workers like Scheie *et al*[38], McLean *et al*[39] and several others. It is believed that ACTH, through its production of cortisone, inhibits the pathological changes in early retrolental fibroplasia, *i.e.*, it decreases the subretinal œdema by decreasing capillary permeability, and it inhibits new vessel formation on retina and in vitreous. Recently, Bembridge *et al*[25] and Brown and Corner[15] have reported satisfactory results with ACTH in early cases. They also opine against excision of the eye as it is found that the child often retains some remnant vision to avoid obstacles in spite of gross intraocular damage.

The disease has to be tackled by a concerted co-operation between the obstetrician, the pediatrician and the ophthalmologist. The obstetrician should make every effort to prolong the pregnancy until the fœtus has attained a weight of more than 4½ pounds (34-35 weeks of gestation), of course, taking into account the welfare of the mother. The pediatrician should exercise great care and strict supervision (for the first six months) in the rearing of a premature baby. Oxygen should be given only when necessary to maintain a good colour and the concentration of oxygen should be maintained at the lowest possible percentage. The ophthalmologist should examine the fundi of all infants weighing 4½ pounds or less, and follow up by subsequent weekly examinations. Reports from workers in India are eagerly awaited.

[36] *Pediatrics*, 3: 824, 1949.
[37] Paper read at Am. Pediat. Soc., 1951.
[38] *Arch. Ophth.*, 45: 301, 1951.
[39] *Tr. Am. Acad. Ophth.*, 55: 565, 1951.

OUR NEW YEAR

The twentyfirst year of the present series of our Journal was completed in September 1952. With the present issue it enters upon a New Year, the twenty-second year of its life.

On an occasion like this, it is customary to review the past and present a programme for the future. We do not want to resolve on a big programme assuring a glorious future. Our Journal, since its inception has been marching ahead. In the present year, we can only assure that our efforts to maintain the standard already acquired, will never slacken.

We are grateful to our Advisory Committees both local and central for their constant guidance in conducting the Journal. We must also mention with thanks the valuable services, received from our learned Referees in the scrutiny and selection of articles and lastly we must express our gratefulness to our numerous contributors for the co-operation they extended by contributing articles for our Journal. It should be acknowledged that a major share of the reputation of our Journal is due to them. We earnestly seek their co-operation in the present year also.

While writing on a similar occasion last year, we announced our intention to convert our Journal into a fortnightly publication. But our plan was thwarted due to some unfavourable factors beyond our control. One of them was the very irregular and undependable supply of paper for printing. The paper position has further deteriorated and we do not see any chance of improvement in the near future. The idea however has not been abandoned. As soon as better conditions are ushered in, we shall look into this question over again and try to implement our wishes of last year.

We also announced last year, the publication of a lay health journal from January 1952. In this respect we have become successful and YOUR HEALTH is being published as a monthly journal from January 1952. We are happy to find that this journal is becoming more and more popular amongst the general public for whom it is meant.

Before we conclude we crave the indulgence of our readers and well-wishers for any shortcomings which might have crept in during the year under review. We solicit the valued co-operation of all members of the I.M.A. and the readers of our Journal far and near for unqualified guidance and support without which we confess it would be difficult to steer the wheel of progress of our Journal.

Wishing very well of all of our friends let us pray that the year we are marching into will be more useful and we shall be able to render a further good account of ourselves.

CURRENT MEDICAL LITERATURE

Fatalities in Insulin Therapy of Psychosis

Scheflen and other (*Arch. Neurol. and Psychist*, 63: 32, 1952) presented eight fatal cases of insulin therapy is psychiatric practice and discussed factors which may contribute to the fatal outcome.

The following considerations may be of value in anticipating adverse reactions: (1) a history of such atypical responsivity to medication as aspiratory collapse or syncope; (2) factors adversely affecting the physiological systems regulating insulin-glucose relationships, such as malnutrition, avitaminosis, use of certain drugs, autonomic imbalance, and excessive fear of treatment; (3) undue resistiveness to insulin as manifested by high dose requirements or the occurrence of relatively few comas. This may be accompained by overactivity of the sympathetic nervous system, excessive fear of treatment, and a tendency to sudden cardiorespiratory complications; (4) undue sensitivity to insulin as manifested by (*a*) low or rapidly decreasing dose requirements; (*b*) unusually deep coma; (*c*) difficulty in termination of coma; (*d*) a tendency to "after-shock," and (*e*) probably, the presence of low vital signs.

Purpura and Pancytopenia Complicating Arsenotherapy

Nelson (*Brit. M. J.*, 1: 300, 1952) reports four cases of blood dyscrasia resulting from the toxic effects of organic arsenicals.

One case of toxic thrombocytopenic purpura recovered spontaneously.

The three other cases showed a pancytopenia with marrow aplasia and ended fatally.

The administration of dimercaprol (B.A.L.) to two of the cases had no apparent effect on the severe toxic manifestations.

Rheumatoid Arthritis

Wiesel and others (*Am. J. Med. Sc.*, 222: 243, 1952) write that inactivation of œstrogenic hormones by the liver is inhibited by the addition of para-aminobenzoic acid. There is a great similarity in structure of cortisone and the œstrogens. The major portion of parenterally administered cortisone is inactivated in the body. These facts made it plausible to attempt to inhibit the destruction of cortisone by the simultaneous administration of para-aminobenzoic acid. The results obtained in the first 15 cases treated by this method are described. Each patient was used as his own control. All had moderately severe or severe rheumatoid arthritis which had previously proved refractory to other forms of treatment.

The two methods used for study were:

1. Patients were started on full-dosage schedule of cortisone acetate until maximum relief of the symptoms of arthritis was obtained. This was usually for a period of 1 week. The daily intramuscular dose of cortisone was then reduced arbitrarily to 25 mgm. On this dose all of the patients developed prompt relapse with severe subjective and objective symptoms and signs within a short time. When this relapse was established, the sodium salt of para-aminobenzoic acid was given orally in doses of 1·5 gm. every 2 hours during the waking period for a total of 8 doses of 12 gm. per day, in addition to the single daily intramuscular injection of 25 mgm. of cortisone acetate. When relief of arthritic manifestations was again established and maintained for variable periods of time control periods were introduced by the substitution of a placebo for the cortisone.

2. Other patients were started immediately on a single daily intramuscular dose of 25 mgm. of cortisone acetate, and 12 gm. of sodium para-aminobenzoate were given orally in 8 divided doses of 1·5 gm. at 2-hour intervals during the waking period. When relief of arthritis was well established, control periods were introduced by replacing the cortisone acetate with placebo injections.

The first method was used in 9 patients, the second in 6 patients. The results, obtained were comparable with those obtained by the administration of much larger doses of cortisone alone, with the additional advantage of a reduction in reactions. The additive or synergistic action of these two compounds and the success obtained in 15 cases warrant further and more extensive use of this method in rheumatoid arthritis.

Nitrogen Mustard in Hodgkin's Disease

Gellhorn and Collins (*Ann. Int. Med.*, 35: 1250, 1951) in giving a critical evaluation of treatment of Hodgkin's disease with nitrogen mustard write:

The course of the disease has been compared in two groups of patients: 67 cases who received radiation and nitrogen mustard in alternating therapeutic courses as indicated, and 65 cases who received radiotherapy alone. The available evidence shows that the four-year survival rate from the onset of symptoms, from the time of biopsy diagnosis, or from the time of evident generalized disease is not significantly different in the two groups of patients. It is concluded, therefore, that the addition of nitrogen mustard to the therapeutic regimen of radiation does not modify the duration of life expectancy of the disease.

Evidence has also been accumulated which demonstrates that the amount of time necessary for radiation treatments is reduced by one-half in the patients receiving radiation and nitrogen mustard when compared to the radiation treatment-days of patients receiving radiotherapy alone. This suggests that the amount of radiation required is less, the asymptomatic period greater and the economic burden lighter for the patients on the former therapeutic regimen than on the latter.

The incidence of serious toxicity in the 144 courses of nitrogen mustard given to the 67 patients was 7 per cent. One death occurred in a patient due to a miliary dissemination of pulmonary tuberculosis following the administration of nitrogen mustard.

Immediate subjective and objective improvement followed 78 per cent of the courses of nitrogen mustard.

It is concluded that nitrogen mustard is a useful adjunct to radiotherapy in the management of Hodgkin's disease in that it reduces the requirements for radiation; however; life is not prolonged by the addition of chemotherapy to the conventional therapeutic regimen for Hodgkin's disease.

Management of Lung Abscess with Antibiotics

Drake and Sons (*Ann. Int. Med.*, 35: 1218, 1951) from the analysis of forty-five proved cases of lung abscess observed as in-patients during the past 10 years observe:

Three conditions considered necessary for the formation of a lung abscess are infection, bronchial obstruction with

resulting atelectasis, and impairment of the arterial circulation to the segment involved.

Predisposing clinical states may be divided into four groups: Those conditions resulting in loss of protective pulmonary reflexes and thus favoring aspiration; local situations in the mouth or upper respiratory tract favoring infections; acute pulmonary pathology, and certain miscellaneous conditions including diabetes, alcoholism and some forms of heart disease.

Two presenting complaints which have heretofore received insufficient emphasis, namely, hæmoptysis and localized chest pain, are emphasized and the variability of the physical signs is cited, while the place of roentgen-ray and the bronchoscope as diagonostic tools is discussed.

It is believed that the importance of dividing cases into a putrid and a nonputrid group, according to the organisms isolated and the clinical character of the sputum, has been overemphasized, since the quality of the sputum is merely a reflection of the degree of obstruction to drainage existing at the moment. It is important to rule out the presence of tubercle bacilli, especially in upper lobe cavities, which formed 65 per cent of our group.

The cardinal printiple in therapy should be establishment of drainage, and where this is not accomplished all other therapeutic efforts will be doomed to failure. Penicillin in large doses should be employed in all cases, but the chief usefulness of this antibiotic will be aborting cavity formation during the "pre-abscess" stage. For this very reason, the true value of penicillin may be difficult to prove statistically but may rather be shown by the decreasing incidence of lung abscess as a complication in respiratory disease treated with antibiotics. Surgery should not be withheld if frequent progress films fail to reveal consistent diminution of cavity size in addition to clinical improvement. After six weeks, complications including fibrosis and bronchiectasis will make serious inroads on the functional efficiency of the involved lung segment, and it seems logical to assume that lobectomy will enjoy increasing popularity. In those cases in which surgery is delayed, the increase in mortality and morbidity will be significant.

Terramycin in Pneumonia

Jackson and Others (*Ann. Int. Med.* 35: 1175, 1952) give in the following the summary of the observations on the treatment of 91 cases of pneumonia.

Clinical and bacteriologic findings in 91 patients with pneumonia who were treated with terramycin have been presented. The bacteriologic results and the immediate clinical effects indicated that terramycin was highly effective against pneumococcal pneumonia. In most of the other pneumonias in which a bacterial etiology was proved or suspected, and in the four cases of primary atypical pneumonia, the immediate effects of terramycin therapy were also favorable.

Replacement of the flora of the sputum by staphylococci, slow clearing of the lungs, and recurrence or persistence of leukocytosis indicated that the beneficial effects of terramycin were, in part, vitiated in many of these patients by superinfections with the staphylococcus.

Gastrointestinal symptoms from terramycin were frequent and in many patients were quite severe. In many of the patients with terramycin-induced diarrhœa, the fæcal flora was replaced by pathogenic staphylococci. These staphylococcal infections of the intestinal tract added considerably to the morbidity of many of the patients who recovered.

Seven of the patients died. In five of these seven patients, the toxic effects of terramycin on the gastrointestinal tract may have contributed in large measure to the fatal outcome. In addition, staphylococci played an important pathogenic role in the lungs of four of these patients.

Antagonism of Penicillin and Aureomycin

Lepper and Dowling (*Arch. Int. Med.*, **88**: 489, 1951, *J. A. M. A.*, **147**: 1713, 1952) write that among 43 patients with pneumococcic meningitis treated with massive parenteral doses of penicillin, 13 (30 per cent) died. Among 14 similar patients treated with aureomycin in addition to the same dose of penicillin, 11 (79 per cent) died. To assure random sampling, alternate patients were selected for treatment with penicillin alone and for treatment with penicillin and aureomycin combined. There was no essential difference between the two groups with respect to age, the number of organisms in the spinal fluid, mental status, portal of entry, pneumococcic complications, the presence of other diseases, or the day on which therapy was started. It is concluded that the poor results with the penicillin-aureomycin combination were not due to the chance inclusion of more severely ill patients in this group, nor did toxicity to aureomycin seem to account for the results. Rather, the evidence indicates that penicillin and aureomycin are mutually antagonistic when employed together in the treatment of pneumococcic meningitis. It appears unwise to use combinations of penicillin and aureomycin, terramycin, or chloramphenicol in treatment of patients with pneumococcic meningitis, or in treatment of any patients, even those with milder infections, in which antagonism has been shown *in vitro*. Consistently good results have been obtained in the treatment of pneumococcic meningitis with doses of 1,000,000 units of penicillin at two-hour intervals without use of the intrathecal route. Since good results have been obtained by the use of aureomycin therapy alone in pneumococcic meningitis, further studies should be made on the efficacy of aureomycin, terramycin, and chloramphenicol, without adjuvant penicillin.

CURRENT TOPIC

NAPT THIRD COMMONWEALTH HEALTH AND TUBERCULOSIS CONFERENCE

The third Commonwealth Health and Tuberculosis Conference organised by the National Association for the Prevention of Tuberculosis was held at the Central Hall, Westminster, from July 8th to July 11th inclusive. Over 800 representatives from 51 countries were present: doctors, hospital administrators, public health officials, nurses, social workers and private individuals.

The Duchess of Kent, President of the NAPT attended the Conference on the first afternoon. In her speech she referred to the progress which had been made in the past few years, and mentioned two problems which, if they could be solved, would do much to assist the control of the disease in Great Britain: the grave shortage of houses, and the lack of nurses trained in tuberculosis work.

The world-wide nature of the problem was emphasised in the first session: Tuberculosis—A Problem for All Peoples. The Duchess of Portland, Chairman, said that

tuberculosis was one of the most vital subjects in preventive medicine: in Great Britain more than 25,000 people died of the disease every year and in India about half a million. It was a problem of civilisation, but what civilisation had caused, civilisation could remedy.

The session was opened by Commander T. D. Galbraith, Joint Parliamentary Under Secretary of State for Scotland, and its keynote was the importance of prevention. As Dr. Brink, from Canada, said: "If there is no tuberculous infection, there can be no disease," and this theme was emphasised by speakers from many different countries: by Dr. Benjamin from India; Sir Andrew Davidson, Chief Medical Officer of the Department of Health for Scotland, who stressed the importance of the education of the public in prevention; Dr. J. B. McDougall, of the World Health Organisation, who advocated an all-out attack on the "black spots" in every region; Dr. Aziz of Pakistan and Dr. Retief of South Africa, who described the Witwatersrand Native Labour Association's arrangements for x-raying new entrants to the industry to the number of 300,000 a year at a rate, when required, of 800 an hour. A most interesting paper was read by Professor Neubauer, of Jugoslavia, who said that before 1940 not much had been done in his country. There were then only 40 dispensaries and 2,880 hospital and sanatorium beds, while in 1951 there were 197 dispensaries—all with x-ray machines and 13,000 beds. Between 1948 and 1950 a B.C.G. campaign had been carried out with international help, and B.C.G. vaccination was now compulsory for all persons up to 25 years of age. It was interesting to hear that mass x-ray examinations are very popular with the Jugoslavs, and attendance is very high—up to 99·4 per cent!

Miss Elizabeth Cockayne, Chief Nursing Officer, Ministry of Health, dealing with the shortage of nurses in Great Britain—a shortage which is paralleled all over the world—said that although there had been an increase since 1948 in the number of tuberculosis nurses and staffed beds, there were still 2,300 unstaffed beds needing 2,600 nurses. It was therefore of the greatest urgency that every effort should be made to prevent the spread of the disease both amongst the public and amongst nurses themselves. Miss Cockayne spoke of the wonderful work which is being done for tuberculous patients, and made a plea to nurses to recognise the interest and importance of this type of nursing and to sanatoria to provide a really good standard of training and experience for their nurses. This last point was stressed very emphatically on another occasion by a health visitor who appealed to doctors to explain difficulties and discuss treatment with their nurses. Nurses did not mind extra work if they could share in the new developments in the tuberculosis field.

Dr. Newing of Australia gave figures with regard to tuberculosis in his country and said that Australia presents the most favourable opportunity in the world for the eradication of tuberculosis. Further details of the control of the disease in Australia were given in a later session by Dr. G. H. Moore, Chief Medical Officer, Australia House—a particularly interesting point being the allowances for tuberculosis sufferers, which were more generous than those in any other country. Speaking from the floor, Mr. Donald Barlow told of his recent visit to Ceylon at the invitation of the Government to investigate the tuberculosis problem there, which was a serious one. The Government had agreed to the following measures: a basic living wage, children's allowances, an active housing programme, an 18-months' subsidy for all tuberculous patients, and the notification of tuberculous cases. He was sure that Ceylon was going to take its place as one of the leaders in coping with the disease.

The second session dealt with DOMICILIARY MANAGEMENT OF THE OPEN RESPIRATORY CASE AND OF THE MINIMAL LESION and with PROCEDURE AT CHEST CLINICS AND LIAISON WITH THE FAMILY DOCTOR. It was felt that home treatment was only a second choice to hospital treatment—Dr. William Brooks, of St. Mary's and Brompton Hospitals, said that to leave children and adolescents living at home with an infectious case was to invite disaster—but that there was much that could be done at home for patients who, in view of the bed shortage, were awaiting admission, or for ex-patients who were still infectious. Dr. Radcliffe, a general practitioner from Wivenhoe, spoke of the vastly improved co-operation between chest physician and family doctor, and said that he had been able to give modern treatment to patients waiting for beds, who in some cases improved so much that admission was no longer necessary. Dr. W. H. Tattersall, Consultant for the Bournemouth area, speaking of the large number of ex-patients who were still infectious, said that once they had been educated in the tuberculous way of life they could resume suitable work without danger to others, and benefit both themselves and the community. Dr. Tattersall gave some interesting results of case-finding in the area. "Case-finding," he said, "is the horse that pulls the cart of the whole tuberculosis control programme," and the most fruitful fields were general practitioners' patients with any kind of respiratory symptoms, contacts of all diagnosed cases and contacts of tuberculin positive children. A most excellent paper on the part the family doctor could play was given by Dr. E. M. Dimock, a general practitioner from Broxbourne. It was a most important part, because tuberculosis was so much a human and a family problem, and the family doctor who was a privileged friend had the opportunity of training his patients in a responsible attitude to both health and disease. Dr. Dimock said that an early diagnosis transcended everything else the family doctor could do for the tuberculous patient (a point again emphasised by Dr. Marc Daniels in the discussion on the new drug, isoniazid). He stressed the importance of personal consultations with chest physicians at which the patient should be present—and of courses for practitioners on new developments in tuberculosis. He welcomed the policy now adopted by some Medical Schools of sending senior students to family practices for a short time as observers. Dr. J. L. Livingstone, Brompton and King's College Hospitals, in his summing-up confirmed the importance of early diagnosis, and said that the undiagnosed case could be more dangerous to others than the known open case. He suggested a place of residence in each town for the "feckless" cases. The good general practitioner was, he said, the backbone of the profession, and in tuberculous cases a knowledge of the psychology and background of the patient was of the greatest importance.

On Wednesday, July 9th, the morning session was concerned with PREVENTION AND CARE WORK BY LOCAL HEALTH AUTHORITIES and with PROTECTIVE VACCINATION. The session was opened by Mr. Iain Macleod, Minister of Health, who gave some figures showing the striking progress made in the campaign against tuberculosis in recent years. In 1951 the number of deaths from the disease was the lowest on record—275 per million against 1,100 in the first decade of the century—and the rate of progress was getting faster. In the last three years the reduction in tuberculosis mortality was as much as one third. Over 6,000 more beds had been made available for tuberculosis treatment since the National

Health Service began, and the number of patients waiting for beds had been reduced from 11,000 in 1949 to about 7,000. But these figures gave no ground for complacency, for there was now on the horizon a possibility of success which would only have seemed a dream to the founders of the NAPT. Referring to B.C.G. vaccination, Mr. Macleod said, "It is not yet felt that the time has come to make the vaccine available for general or indiscriminate use among the population at large. This view, though it may be thought unduly restrictive by some, is based on responsible expert judgment, and is not due to limitations of supply of the vaccine or to financial economy. Neither is it necessarily a fixed policy. My Ministry will take careful account of whatever expert advice may be forthcoming in the light of continuing study and research."

Dr. J. A. Scott, Medical Officer of Health, London Country Council, gave an interesting account of the position of London in the field of tuberculosis. The risk of infection was considerable owing to crowded transport, crowded public places, and bad housing conditions, but diagnosis by mass x-ray was good, the findings being lower than for the country as a whole, and the tuberculosis services were well organised. Dr. Scott gave it as his opinion that mass radiography should be in the hands of local authorities, and not regional hospital boards, as it was a preventive and social measure. He ended by saying that tuberculosis was now in a definitely hopeful condition, and that one of the big present day problems was how to use ex-patients in industry. Alderman Yorke, of Sheffield, made a valuable contribution to the session. After referring to the work done by the local health authority in Sheffield before the National Health Service Act, when tuberculosis mortality was reduced during the first half of the century from nearly 3 per 1,000 to just above 0·3 per 1,000, he spoke of the fear of tuberculosis leading to delay in treatment and lack of preventive measures. The importance of health education through the informal talks of the health visitor and among adolescents by every available means, such as youth clubs, night schools, Sunday schools and churches, could not be over-emphasised. Fear was again the reason why the general public did not make enough use of the mass x-ray services. Some interesting papers followed dealing with care and after-care, in which special reference was made to the housing problem and to team work in rehabilitation. A comprehensive resume of the work achieved in Northern Ireland by the Tuberculosis Authority established in 1947 was given by Alderman Andrew Scott, Vice-Chairman.

Papers on B.C.G. vaccination were read by Dr. Marcia Hall, of Great Ormond Street Children's Hospital, London, who described methods and immediate results; Dr. van Diense, of the Pasteur Institute, Paris, who gave particulars of what was being done in France, where such vaccination was made compulsory in 1950, and by Dr. Margaret Macpherson, of Brompton Hospital children's contacts department, who described the research work being done there. Dr. van Diense also mentioned the large-scale voluntary campaign carried out by UNICEF in the French Overseas Territories: Morocco, Algeria, and Tunis, from 1949 onwards, when over two million people were vaccinated. In summing up, Dr. Greenwood Wilson, of Cardiff, made the interesting suggestion that the help of shop stewards should be enlisted in dealing with the problem of tuberculosis in industry.

The Wednesday afternoon session on CONTEMPORARY IDEAS IN THE MANAGEMENT OF THE TUBERCULOUS PATIENT was opened by Dame Dehra Parker, Minister of Health and Local Government, Northern Ireland. In addition to telling the Conference what was being done in Northern Ireland to-day, Dame Dehra referred to the problem as a whole, and said that publicity on the progress made, and education were both vitally important—the one as a tonic to the patient, and the other as the best means of prevention. Mr. Holmes Sellors, Thoracic Surgeon, Middlesex Hospital, spoke of the place of the surgeon in tuberculosis. He referred to the proper timing of surgical intervention, a most difficult thing to decide. Mr. Holmes Sellors discussed the recent advances in chemotherapy and in excision. On the surgical side he favoured thoracoplasty unless impracticable. Two important and interesting papers followed: one by Dr. Ralph Tompsett, of the Cornell Medical School, U.S.A., and one by Dr. Marc Daniels, of the Medical Research Council, both dealing with the new drug, isoniazid, and both pointing out that as tuberculosis destroys tissues, drug therapy cannot eradicate tuberculosis but can only prevent further destruction. The importance of early diagnosis and treatment cannot therefore be over-stressed, thus enabling greater use to be made of chemotherapy, and in the same way (to quote Dr. Daniels): "rest, good food, collapse therapy, surgery, all continue to be fundamental." Dr. Tompsett described the researches at Cornell under Dr. Walsh McDermott in the use of isoniazid in cases of miliary and meningeal tuberculosis, and said that there had been marked and persistent improvement, particularly in the former group. He stated that observations must be carried out on far larger numbers of patients for much longer periods of time, and that study of the question of drug resistance, which is likely to be a limiting factor, is also necessary. But isoniazid was yet another valuable weapon in the campaign. Dr. Marc Daniels mentioned the successes obtained with new drugs such as streptomycin and P.A.S. in cases of miliary and meningeal tuberculosis, and said that before their discovery these forms of the disease were almost uniformly fatal. It was obviously worth trying any new drug which was effective in cases which did not respond to the present drugs, or were intolerant or became resistant—and that isoniazid was effective was clear from Dr. Tompsett's information. Dr. Daniels described the Medical Research Council trials of isoniazid, which started in this country about four months ago, and said that eleven hospital regions and thirty-eight hospitals were involved, as well as chest physicians in scores of clinics in England and Scotland. Isoniazid was being compared with streptomycin plus P.A.S. and was also on trial when used in combination with streptomycin.

The next speaker was Dr. Houghton of Harefield Hospital, who was convinced that the tuberculous patient needed specialised medical, surgical and nursing attention in a hospital for many months. This view was strongly criticised by Dr. Peter Stradling, of Hammersmith Hospital, who felt that domiciliary treatment was essential in view of the lengthy waiting period for sanatorium beds. Dr. Houghton said it was important that the patient should be appreciated as an individual, and his resistance encouraged in every way. Dr. Clausen of Vordingborg Sanatorium, Denmark, also spoke of the encouragement of resistance in his address on tuberculosis amongst children in Denmark: a few years ago there were waiting lists for children's sanatoria, now there were many free places, and a first batch of British children has gone over this year. There were one million children in Denmark, and in 1951 only 25 died of tuberculosis, *i.e.*, 2·5 per 100,000. Good food, occupational therapy, such as handicrafts, and an educational curriculum when possible

were all part of the plan for improving natural resistance. Dr. H. J. Hambling, Sir Halley Stewart Research Fellow, spoke of the psychological side of tuberculosis. He said that emotional worries lowered resistance, thus making people more prone to the disease, and that the emotional shock caused by a long and disabling illness such as tuberculosis prevented recovery. Co-operation between the chest physician and the psychiatrist was long overdue. This very valuable session was summed up in an able and most entertaining way by Mr. d'Abreu, of Birmingham Regional Hospital Board.

On Thursday, July 10th, the Conference considered the question of "Tuberculosis in British Colonial Territories," and a very broad view of the subject was obtained from the papers given by doctors from many parts of Africa, the West Indies and elsewhere. The session was opened by Mr. Oliver Lyttelton, Secretary of State for the Colonies, who said that since malaria had been brought under control tuberculosis now headed the list of killer diseases in these territories. Its eradication was not only a medical matter, but depended on action by all members of the community, and Mr. Lyttelton said he wished to pay generous tribute to the NAPT for the help it had given, especially in arrangements for colonial doctors to study the latest methods at home, and for planning schemes for tuberculosis control in colonial territories. Mr. Lyttelton also gave some particulars of progress achieved in various places, such as Jamaica, Aden, Hong Kong, Singapore and the South Seas. Dr. Pridie, Chief Medical Officer, Colonial Office, said that tuberculosis was a serious problem, but that its seriousness was now appreciated, and also referred to the assistance given by the NAPT in this respect. He spoke of the devoted efforts being made by the medical profession, the public and the international health organisations, and said, that the disease in colonial territories was a local problem which had to be dealt with according to local factors, but that each territory could learn from the others and especially from all the expert knowledge and experience in Great Britain. This was why the present Conference was so valuable, because here was pooled knowledge from all over the world.

Dr. Briggs described the anti-tuberculosis plans in Northern Rhodesia, the important point about which was that the service had been started before the country become industrialised, thus affording an opportunity of demonstrating that tuberculosis was preventable. He also said that the African had probably more natural resistance to tuberculosis than had been supposed. This point was also confirmed by Dr. Vincent Hetreed, who is undertaking a tuberculosis survey in Nigeria. Further papers dealt with tuberculosis in British Guiana, Nyasaland, Mauritius, Fiji and the High Commission territories of Bechuanaland, Basutoland and Swaziland. Two interesting addresses on the position in Singapore were given by Dr. G. H. Garlick of the Singapore Anti-Tuberculosis Association and Dr. W. J. Vickers, Director of Medical Services, Singapore. There was no doubt that tuberculosis was a serious menace in Singapore, and the mortality and incidence figures were difficult to assess. It had been estimated that the mortality rate was about 105·2 per 100,000, and the incidence rate below 2 per cent, but Dr. Garlick, whose views were considered by Dr. Vickers as rather gloomy, put both much higher, and estimated the incidence as more like 8 per cent. Energetic control measures had been put in hand. The Government programme allowed for two hospitals with 1,100 beds, 500 of which were now available, a complete out-patients scheme, including B.C.G. vaccination—at present there was a Government clinic, with x-ray facilities—domiciliary relief, the first of its kind in that part of the world, and improved housing conditions. Magnificent work had been accomplished by the voluntary anti-tuberculosis association. In view of the large-scale nature of the problem, ambulatory treatment was the only possible course, and a new clinic was built—the cost being raised by voluntary contributions—with complete diagnostic and treatment units. The results had been surprisingly good, and it was quite amazing that they could be obtained with patients who were very poor, lived in grossly over-crowded conditions were under-nourished and must go on working hard during treatment. The explanation appeared to be the high natural immunity of the Chinese to tuberculosis, which if it did not bring the patient too late for all treatment, led to rapid recovery even under ambulant conditions.

Professor F. R. G. Heaf, in summing up this session said that the reports showed what remarkable work was done in the Colonies considering how isolated the medical officers were from the facilities available in this country. He emphasised the value of careful research and careful investigation by the man on the spot, as the problem varied from colony to colony. The question of the resistance of the native population to tuberculosis and of improving this by good living conditions was important. Prevention must take prior place, but treatment could not be neglected.

The Chairman of the session on Non-respiratory Tuberculosis including Bovine Infections and their Results was Sir Robert Young, Vice-Chairman, NAPT Council. In introducing the session Sir Robert said that he had seen so many sad cases of tubesculosis of these types that he welcomed a discussion on the subject. Dr. Charles Hill, Parliamentary Secretary to the Ministry of Food in discussing the question of Bovine Infection, spoke of the two parallel lines of attack: the longterm policy of the eradication from herds of reactors to tuberculin testing, and the short term policy of making milk safe by pasteurisation and sterilisation. Dr. Hill said that with regard to the elimination policy, it might not be long before areas were designated as eradication areas, and that in the meantime one third of the total number of cattle were now attested, and 80 per cent of milk was heat-treated. My 1959 almost the whole country would be covered by the Special Designations Act, and from 1954—a most important point—pasteurisation would include bottling and sealing where pasteurised, thus avoiding all human infection. He was very glad that to-day the old controversies of eradicating versus pasteurisation and of heat-treated versus raw milk had died down—it was now agreed that there were diseases conveyed by milk which were preventable and therefore should be prevented, and the only problem was whether the rate of progress was fast enough. Professor Selby Wilson read a most interesting paper, putting the whole subject of Bovine Infection in its historical perspective. He recounted the great struggles of the late 19th and first half of the 20th century to prove first, that tuberculosis of the bovine type could occur in man: 'the cow was one of our worst enemies as well as one of our best friends', and, secondly, that heat-treated milk destroyed infection but not nutritive value, a struggle which was ended by the compulsory pasteurisation act of 1948. Mr. Harold Woolley, of the National Farmers' Union, dealt in a very practical way with the considerations involved in building up a tuberculosis-free herd and eradicating reactors. He discussed questions of expense, buildings, testing—which was not popular with the cows—and said that he was convinced that in the long run the eradication of tuberculosis from herds would prove a sound investment from an

economic standpoint as well as from that of public health.

After the discussion on Bovine infections papers were read by specialists on the different forms of non-pulmonary tuberculosis: Orthopaedic, genito-urinary, ocular and skin tuberculosis and on tuberculosis associated with diabetes. Mr. Evan Evans, Medical Superintendent of the Treloar Hospital, discussed the orthopaedic type and Mr. J. H. Carver, in a most valuable contribution on genito-urinary tuberculosis, stressed the importance of three points in order to improve on the present depressing figures for nephrectomy: a 50 per cent mortality within 5 years. These points were not to regard renal tuberculosis as an isolated lesion, to appreciate the significance of the early symptoms and to give careful and prolonged after-care. Mr. A. G. Cross discussed ocular tuberculosis, and gave an account of the work at White Oaks, the first Eye Sanatorium in the country, where satisfactory results were being obtained, largely through rest therapy. The paper prepared by Dr. F. S. Airey, of Malaya, and read in his absence, recounted the passing of the problem of skin tuberculosis, a most disfiguring and horrific form of the disease, and added a warning against failing to diagnose it, now students had few opportunities of observing cases during training. Dr. Luntz of Romsley Hill Sanatorium, Birmingham, read an interesting paper on the tuberculous diabetic, and said that there were over 3,000 of these cases in the country. Special treatment for both diseases simultaneously was needed, but the Romsley Hill Sanatorium at Birmingham was the only centre of the kind in Great Britain at the present time. Expense must be considered, but existing wards in hospitals could be adapted.

The last morning of the Conference was devoted to Social Welfare, the subjects being THE SOCIAL WORKER AND THE TUBERCULOUS FAMILY and THE PATIENT IN INDUSTRY. The chair was taken by Dr. R. R. Trail, Medical Director, Papworth Village Settlement, who said that tuberculosis affected every aspect of the patient's life and that home conditions were of the greatest importance. Dr. Trail said that one of the biggest steps forward in recent years was the Ministry of Labour's Training Scheme for Disabled Persons Act. "We fail completely," he said "unless the patient can be returned home and to industry as a self-respecting member of the community." In the first section a lively and provocative speech was made by Dr. Frank Ridehalgh, of the Churchill Hospital, Oxford, who said that the nation could not afford the cost of tuberculosis, especially from the point of view of loss of production. Dr. C. L. Sutherland, Ministry of National Insurance, gave an account of tuberculosis in the national insurance scheme. Alderman Colton, from Northamptonshire, spoke of the work of the voluntary care committee: 'the added jewel to the crown of all the work which was being done', and said that its importance was that it was really down to the patient's level.

The section on "The patient in Industry" dealt with the subject in a very comprehensive way and it is interesting to note that representatives of over 30 commercial firms attended the Conference. The first speaker was Dr. J. O'Dwyer, Medical Officer, Lever Bros. and Unilever, Ltd., who said that industry could help in dealing with tuberculosis by the early discovery of cases with infection, by finding suitable employment for patients after treatment, or while under treatment—though the number of light jobs was very strictly limited—and by health education. Mrs. Nora Guest, of Remploy Limited, gave an account of the 92 Remploy factories, 7 of which were for tuberculous cases requiring special conditions, though quiescent and surgical cases were employed in the normal factories. Miss Mary Grainger, of the Disabled Persons Branch, Ministry of Labour, described the work of the Ministry's resettlement officers, and said that about 1,200 tuberculous patients had completed courses at industrial rehabilitation units during the past 3½ years, of whom 80 per cent had been placed in open employment or training. Miss Grainger referred to the valuable work of village settlements for tuberculous persons, a point which had been emphasised already by the chairman. The Rev. William Bodin, Social Relations Officer of Stewart and Lloyds, Corby, referred to the miracle of the last half century: the completely new conception of tuberculosis as a disease, like many others, which could be prevented and could be cured. "Medical science", he said, had lifted tuberculosis out of its grim isolation and exclusiveness, and the greatest need of to-day was "to spread abroad the good news of the great progress that has revolutionised the whole field since the war, so that everywhere in industry, in society, in home life, there will be an increasing confidence that comes from a real sense of hope." Dr. Edens, of Philips Electrical Limited, gave a resume of the measures against tuberculosis taken by this firm in Holland. Special stress was laid on case-finding, B.C.G. vaccination and rehabilitation, and the death rate in the factory employees in 1951 was 3·5 per 100,000 as against 16 amongst the general population. Dr. Peter Edwards, of the Cheshire Joint Sanatorium, in summing up said, "the return to work is the vital part of the doctor's job to the patient".

A new feature of this Conference was the holding of special group meetings for those with particular interest. For doctors there were demonstrations on laboratory technique, a radiological demonstration and a discussion on modern technique in diagnosis and in addition a joint meeeting with the British Tuberculosis Association on Child-bearing and Tuberculosis. For sanatorium matrons and nurse teachers there was a discussion on the Protection of Nurses against Tuberculosis. The meeting for social workers and health visitors to discuss the Fabric of Social Services to meet the Patient's needs stressed the importance of team work by regular meetings at chest clinic or health centre of all concerned with the medical and social care of the patient. Another important discussion was on the Housing Problem of the Tuberculous Family. Tuberculosis gave housing priority. Good houses for all, rather than building especially for the tuberculosis patient and his family was favoured. It was stated that of the 33,000 tuberculosis people in London, only half had a separate bed-room.

XVII U. P. MEDICAL CONFERENCE

XVII U. P. Medical Conference will be held at Sitapur on the 25th and 26th October, 1952. Members of the profession are requested to make it a success by presenting papers and taking special interest in the Scientific Section.

All inquiries and communications should be addressed to Dr. B. D. Kapur, M.B.B.S., Organising Secretary, XVII U. P. Medical Conference, Health Pharmacy, Sitapur (U.P.).

NOTES AND NEWS

JAPAN MEDICAL ASSOCIATION SENDS MESSAGE OF GOOD-WILL TO I.M.A.

Dr. Takeo Tamiya, President, Japan Medical Association, Tokyo, in a letter dated 8-8-52 sent a message of goodwill to the President of the Indian Medical Association. It runs—

It is our great pleasure to send a message of good-will to you, President of Indian Medical Association, on behalf of all the members of the Japan Medical Association, which is the largest civil organization of doctors in Japan and has the membership of 60,000.

We heartily believe in the solemn ideal of the World Medical Association, which aims at promotion of world peace through our contribution to the development of medical sciences and that of the medical treatments. Our Organization was granted the membership of this world association last year after we had started our work anew in 1947, re-organizing our association of the pre-war conservative character into progressive and up-to-date organization.

Our duty, we believe, is especially grave because we live and should improve the unhealthy condition of the Orient, where epidemics are prevailing wider and oftener than the other parts of the world where public health is kept in better condition.

Our duty lies, we believe, in co-operation of the doctors of various countries of the Orient to make the Orientals more healthy, or, we desire, to promote the hygienic conditions of the Orientals up to the highest standard of the world health condition.

We believe that this is possible only through the mutual co-operation based on the mutual understanding. We desire the peace of the Orient and the happiness of the Orientals would be achieved by the efforts of the doctors through their sincere services.

We hereby ask you your help would be extended to us to the mutual friendship and co-operation of the Medical Associations of both countries, India and Japan, to accomplish our common ideal. I tell you, that my proposal is agreed and supported by all the Japanese doctors who are the members of our Association.

Hoping that all the members of your Indian Medical Association would join this worthy work.

MEDICAL GIFTS FROM GERMANY

"It is not the magnitude of the gift that matters. The quality of the spirit behind it has earned the immeasurable gratitude of our country," said Rajkumari Amrit Kaur, Minister of Health, Government of India, while receiving a gift of medicinal supplies from His Excellency Dr. Ernst Wilhelm Meyer, Ambassador of the Federal Republic of Germany, at a brief ceremony held in New Delhi on August 12, 1952.

The gifts, of a total value of Rs. 1,20,000, consist among other items of PAS Sodium Tablets and Supronal Tablets. The PAS Sodium Tablets are being allocated to Madras and Bihar and Supronal Tablets to Mysore, Assam, Madras, Rajasthan, Uttar Pradesh, West Bengal, Ajmer, Cutch, Madhya Bharat and the Punjab.

WORLD CONFERENCE ON MEDICAL EDUCATION

The first world conference on medical education, sponsored by the World Medical Association, will be held at the British Medical Association House, London in August 24-29, 1953. Themes of discussion will consist of (1) Requirements for Entrance into Medical Studies and Selection of Students; (2) The General Aims and Content of the Medical Curriculum; (3) Techniques and Teaching Methods in Medical Education; (4) Social Medicine—Its Concepts and Place in the Medical Curriculum.

Sir Lionel Whitby, Vice-Chancellor of Cambridge University will preside. The sessions will be both plenary and sectional. There will be prepared papers, selected discussion and general discussion. Throughout the sessions of the Conference, a determined effort will be made to bring out a free exchange of ideas, to develop possible unification of thought, to explore ways and means of assisting underdeveloped countries and to gather the opinions of the practising physicians of the world—the ultimate consumers of medical education.

The Secretary General of the WMA has issued invitations to all member National Medical Associations, the Dean of each medical school and Governments and Organisations affiliated with WHO, IUA and ICMS.

CERTIFICATE AND DIPLOMA IN PUBLIC HEALTH AND DIPLOMA IN INDUSTRIAL HEALTH

The Royal Institute of Public Health and Hygiene, London conducts recognised Courses of Instruction (starting annually in March and October), for the examinations of the Conjoint Board of the Royal College of Physicians of London and the Royal College of Surgeons of England, for the Certificate in Public Health.

These lead to Courses (commencing in January and August) for the Diploma in Public Health, and, similarly, (in February and July) for the Diploma in Industrial Health.

Students are also prepared for the Diploma in Industrial Health examinations of the Society of Apothecaries of London.

Any of the Courses may be taken whole-time or part-time. They consist of both lectures and visits.

Candidates desiring a Course for either the Diploma in Public Health, or the Diploma in Industrial Health of the Conjoint Board, are required by them to produce evidence of being in possession of a recognised Certificate in Public Health.

For the Certificate in Public Health, lectures are given by specialists on the various sociological aspects, and in the Diploma in Public Health, the practical work is carried out at a County Borough.

The Diploma in Industrial Health Course entails visits to all types of factories and to coal mines, etc.

Further information, entry forms and prospectuses may be obtained from the Secretary of the Institute, 28, Portland Place, London, W. 1., or from the Dean, at 23, Queen Square, London. W. C. 1.

NEW ANTI-T. B. DRUG RELEASED FOR SALE

The Government of India have decided to release the new anti-T. B. drug isonicotinic acid hydrazide as well as other hydrazine derivatives of isonicotinic acid for sale to the general public. In accordance with Rule 30-A of the Drugs Rules, 1945, however, importers of these drugs will have to

obtain prior permission of the Drugs Controller (India) Directorate General of Health Services, New Delhi, in writing, for this import. This permission will ordinarily be given subject to the condition that, in addition to compliance with other provisions of the Drugs Rules, 1945, the drugs are labelled with the words: "Caution. It is dangerous to take this preparation except under medical supervision." The sale of the drugs will only be permitted on the prescription of a registered medical practitioner. Import license under the relevant Import Trade Control regulations will also be required for the import of these drugs.

The restrictions regarding labelling and retail sale will also apply to the drug manufactured in India.

HEALTH MINISTER VISITS PROJECT OFFICERS' TRAINING CAMPS

Rajkumari Amrit Kaur, Minister for Health, visited the Project Officers' Training Camp at Nilokheri and discussed with the officers some of the problems regarding the execution of the community projects programme in their areas to be started in October.

The Minister stated that the main object of the programme was to inspire and create leadership in the villages so that the people themselves might be enthused to help themselves to create better conditions of living. She urged on the officers to lay more emphasis on the preventive aspect of medicine and health in the villages.

CORRESPONDENCE

The Editor is not responsible for any views expressed by contributors

STUDIES IN CHOLERA

SIR,—I have read with interest the article "Studies in Cholera" by Rao and others in April 1952 issue of Journal of the Indian Medical Association (**21**: 295, 1952).

From the article it appears that the investigation done is not complete and from the incomplete investigation it is wrong to say that the epidemic was mainly water-borne in the two towns *i.e.*, at Kankanhalli and at Koratagere; from inclusive investigation the authors could not find out the character of epidemic at Bangalore. I think that the following epidemiological observations were necessary to complete the investigation.

1. Firstly at Kankanhalli the cause of occurrence of the first case and subsequent cases and the link between the cases have not been studied.

2. The cases have not been presented chronologically in the map.

3. The assumption that "the cause of this recrudescence may perhaps be due to the fact that some people were using the river as a source of domestic water supply" (page 295), is vague; whether the persons who were using the river water, got only cholera during recrudescence has not been mentioned and there is also no mention whether those people who had been previously inoculated got cholera during recrudescence; the river water was not bacteriologically examined.

4. There is no mention whether there was abundance of the fly nuisance.

5. "A carrier state in cholera is usually discredited (page 302)" and so stools of possible carriers have not been examined; the possibility of fly-borne and carrier state being excluded, Bangalore problem has baffled the authors.

6. At Koratagere similar investigations are lacking except that the river water has been examined during second attack of cholera; water may be infected secondarily by the washings of primary cases.

7. The distances between Kankanhalli, Koratagere and Bangalore and their contiguity have not been mentioned. There is also no mention if there is any transport system between the towns.

8. Bangalore town has been attacked with cholera after Kankanhalli and Koratagere were affected; so whether the first case at Bangalore was an imported one has not been mentioned. One Mrs. A. H. (page 302) confounded the authors but if the carrier state or fly-borne possibility had not been excluded, the problem would have been solved. There is no mention whether she visited any restaurant or brought any food from outside or she took cold food or took the milk cold or kept exposed after the milk was boiled, whether there was a fly nuisance or whether there was any cook and whether the cook was a carrier.

As a rule, longdrawn epidemics like those reported are not waterborne. I am etc

23-6-52,
Gopalganj P. O.
Saran Dist.

SUSHIL K. SAMANTA,
Member of I.M.A.,
Gopalganj Branch.

* * * *

SIR,—I am grateful to Dr. Sushil Kumar Samanta for his criticism of the paper.

As a senior officer was responsible for pulling up, I am answering the points raised in Dr. Samanta's letter. It was not claimed that the study was complete as it was only a prelude to studies to be conducted in other parts of the State where cholera has become an yearly event.

(1) It is the experience of every epidemiologist that the first few cases are never recognised and after a few fatalities have happened he is called to review the cases. With reference to Kankanhalli, it was during the recrudescence after a lull, that the studies were begun. At this stage it was distinctly traced to cases having occurred among people who were allowed to take water from the river. After the recognition that there were cholera attacks during the first 'epidemic' the police were requisitioned to see that no one approached the river for using the water. The police protection was withdrawn during the period when there were no cases. When people started using the river water again, a second series of cases occurred.

(2) As the maps were to be small and there would have been a overcrowding if the dates of the attacks had been marked on the maps, they have not been dated. The blood of the people who survived the second attack had been serologically examined and is forming the subject of another study to be published. There were four cases of attacks among people who had been protected. These people had been interviewed and their blood taken for serological examination.

In the beginning when the so-called gastro-enteritis were recognised to be possible cholera, DDT and Gammexane spraying were taken up on a large scale in Kankanhalli, Koratagere and in parts of Bangalore.

At Koratagere also after the first case had come into the town from outside, the police were used to prevent the water of the river being used for drinking, the people were exhorted to use only cholorinated well water. When there was a subsidence of attacks the Police were withdrawn; the recrudescence occurred in a very few days. It was only because of these events, that it was suggested that the recurrences of cholera cases were waterborne. Because of Gammexane spraying immediately during the first attack the fly nuisance was completely controlled.

(7) Kankanhalli is South of Bangalore at a distance of 36 miles. Bangalore being the headquarters of the Mysore State, transport facilities from every town in the State always exist towards Bangalore.

(8) In June 1951 in Bangalore quite a few cases clinically called gastro-enteritis occurred. It is impossible to state whether these first few cases at Bangalore were imported. However, the first case which was recognised bacteriologically as true cholera was in a Mahomedan house where extremely clean conditions prevailed and the people, educated and intelligent, would not permit normally any variety of infection. It was definitely understood that the water was from the chlorinated City supply and the lady being a gosha lady had no contact with any outside food or drink. It was because of this that it was stated that this case was not understandable. It was also the first recognised case where cholera vibrios were isolated and serologically typed. Quite a few cases had not been sent for bacteriological analysis. It is therefore impossible to say whether the earlier cases that occurred were due to V. cholera.

Further studies are being conducted and will be reported duly.

In June a number of cases which were thought to be cases of gastro-enteritis occurred. I am etc.

4-8-52. C. V. NATARAJAN,
Public Health Institute, *Dy Director of Public Health.*
Bangalore.

REVIEWS

A HISTORY OF NEUROLOGICAL SURGERY—Edited by A. Earl Walker. Pp. XII+583 with 152 figures. Published by Balliere, Tindall & Cox, London. Price 91s. 6d. net.

This is a comprehensive monograph about the origin and development of neurological surgery. Starting from prehistoric times the reader is taken through successive centuries and is given a composite view of the progress of neurological surgery in different countries of the world.

It is easy to visualize how progress of this science has kept pace with the development and fall of the different civilizations in the world.

The pattern of the operative technique in its many details are dealt with as well as the origin of the numerous ancillary diagnostic procedures. A very useful part of the book is the short life sketch of many pioneers in this field—Horsley, Macwen, Cushing and others.

There is an extensive bibliography at the end of the book running to more than 50 pages. This considerably enhances the value of the book and it can be safely recommended that every neurologist and neurological surgeon should possess a copy.

A. K BASU.

CAUSES AND PREVENTION OF TUBERCULOSIS—By Brice R. Clarke, M.D., Published by E. & S. Livingstone, Ltd., Edinburgh and London. 1952. Price 32s. 6d. net.

The book is divided into 15 chapters. The earlier chapters consist of detailed reviews of the most up-to-date information on the method of infection, social and environmental factors, the influence of food, of age, of race, of family etc. In Chapter X the causes of tuberculosis have been summarised and principles for prevention enunciated.

The author points out that there is little disagreement about the facts regarding pathogenesis, epidemiology, clinical course, etc., but considerable differences exist on generalisations deduced from the facts. Scientific preventive medicine depends on a correct understanding and interpretation of the scientifically observed facts. Dr. Clarke has admirably reviewed the available up-to-date information on the pathogenesis and epidemiology and discussed the various preventive measures scientifically in the light of the known facts. The book will be of immense value to all students of Tuberculosis particularly those who are engaged in antituberculosis work, for a correct understanding of the complex problems of epidemiology of tuberculosis and the various preventive measures.

Mass miniature radiography and B.C.G. vaccination have been dealt with extensively in two separate chapters. The author has also rightly called attention to the fact that active political and public support is essential for the success of any antituberculosis campaign and therefore a proper understanding of the social attitude and the "climate of opinion" in the community is necessary.

P. K. CHATTERJEE.

YOUR QUESTION

Correspondents should give their names and addresses (not necessarily for publication) and include all relevant details in the questions which should be typed. Questions of medical interest only will be acceptable for this section. They should be sent in a separate envelope marked. "Your Questions." Questions of general interest will be published in preference. Lack of space precludes answering all the questions received.

Q. A doctor has certified that "the person has consumed *arrack*" by smelling his breath and noting the general appearance. Is it possible to find out the kind of alcohol consumed by smelling the breath alone? If not, how to find out?

Ans. It is not possible to give an opinion on the kind of alcohol or alcoholic drink consumed by a person by the smell of his breath or by his general appearance. Even the estimating of the percentage of alcohol from the washing of the stomach does not help to find out the kind of alcohol drunk as it depends on so many different factors *viz.*, the dilution, the diluent, the presence or absence of food in the stomach, the time which has elapsed since and as so many different drinks have nearly the same percentage of alcohol in them!

ERRATUM

In the Index to Vol. XXI published with the September 1952 issue, the name of Hiralal Saha, the co-author of the Original Article "Frog (*Rana Tigerina*) Test for Diagnosis of Pregnancy", was inadvertently omitted. The article was published under his joint authorship with Chunilal Mukherjee in May 1952 issue, page 335.

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Potassium Glycerophosphate	...	gr. 4
Calcium Glycerophosphate	...	gr. 4
Strychnine Glycerophosphate	...	gr. $\frac{1}{60}$
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SUPPLEMENT

Journal of the Indian Medical Association

VOL. XXII. No. 1 CALCUTTA OCTOBER, 1952

I.M.A. WORKING COMMITTEE

PROCEEDINGS OF THE 44TH MEETING HELD AT INDORE ON 28TH AND 29TH JUNE, 1952

[SUBJECT TO CONFIRMATION]

MEMBERS PRESENT: (a) *Ex-officio members*: 1. Dr. T. S. Tirumurti (Madras), President (in the chair); 2. Dr. A. K. Sen (Calcutta), Senior Vice-President; 3. Dr. S. C. Sen (Delhi), Honorary General Secretary; 4. Dr. A. P. Mittra (Delhi), Honorary Joint Secretary; 5. Dr. R. Sinha (Calcutta), Honorary Joint Secretary; 6. Dr. D. V. Venkappa (Madras) Honorary Joint Secretary; 7. Dr. A. D. Mukharji (Calcutta) Editor, J. I. M. A.

(b) *Representatives from the Provincial Branches*: 8. Dr. A. C. Ukil; 9. Dr. P. K. Chatterjee; 10. Dr. A. K. Bose, (in place of Dr. J. Mojumdar); 11. Dr. K. K. Sen Gupta, (in place of Dr. B. P. Neogy) Bengal Provincial Branch; 12. Dr. B. R. Nayen; 13. Dr. C. S. Thakkar, Bombay Provincial Branch; 14. Dr. P. N. Sinha; 15. Dr. Rajeswar Prasad, Bihar Provincial Branch; 16. Dr. P. R. Trivedi, Gujarat and Saurashtra Provincial Branch; 17. Dr. G. V. Joshi; 18. Dr. B. V. Mulay, Maharashtra and Karnatak Provincial Branch; 19. Dr. Ram Rakha Dheer; 20. Dr. F. C. Shori, Punjab Provincial Branch; 21. Dr. T. S. Balasubramaniam (In place of Dr. T. V. Sivanandam); 22. Dr. P. B. Annangarachari, (In place of Dr. U. Krishna Rau), South Indian Provincial Branch; 23. Dr. R. A. Bhagwat, Madhya Bharat Provincial Branch; 24. Dr. D. G. Ojha, Rajputana Provincial Branch; 25. Dr. S. S. Rao, Travancore-Cochin Provincial Branch; 26. Dr. (Capt.) H. N. Shivapuri; 27. Dr. B. Thungamma (In place of Dr. H. Hukku), Uttar Pradesh Provincial Branch; 28. Dr. T. K. Dayalu, Mysore Provincial Branch.

(c) *Co-opted members*: 29. Dr. P. K. Guha, (Calcutta); 30. Lt.-Col. T. S. Shastry, (Madras); 31. Dr. Chamanlal M. Mehta, (Bombay).

(d) *Invited members*: 32. Dr. D. N. Khandekar, (Indore); 33. Dr. S. K. Mukherji, (Indore); 34. Dr. B. C. Bose, (Indore).

The meeting was scheduled to be held at 9-00 A.M. but since a large number of members from Bihar, Bengal and South India were unable to reach in time because of a temporary suspension of Railway traffic from Khandwa, due to a minor accident, the quorum was not complete. The meeting, therefore, commenced at 2-30 P.M. in the Tagore Library of the Indore Medical College.

In the absence of the President or the Senior Vice-President, Dr. Chaman Lal M. Mehta—a Past President of the Association, was proposed to the Chair.

Item 1: *CONDOLENCE RESOLUTION.*

The following condolence resolution was unanimously passed, all members standing and observing silence for 1 minute:—

"This meeting of the Working Committee of the Indian Medical Association places on record its deep sense of sorrow at the sad demise of the following members of the Association and conveys its heartfelt sympathy to the members of the bereaved families:—

1. Dr. Des Raj Sapra (Kangra Branch); 2. Dr. Ved Prakash Gupta (Meerut); 3. Dr. (Miss) Salima Hector (Dinapore); 4. Dr. (Miss) Nancy Tallon (Dalmianagar); 5. Dr. Guruja Kumar Deb (Gauhati); 6. Dr. K. N. Ramanathan (Coimbatore); 7. Dr. Attauddin Ahmed (Assam); 8. Dr. P. Varadarajan (Madras); 9. Dr. R. Krishna Iyer (Coimbatore); 10. Dr. U. Rama Rao (Madras); 11. Dr. Naoroji Faramji (Ahmedabad); 12. Dr. R. K. Naidu (Poona); 13. Dr. C. R. Kittur (Belgaum); 14. Dr. B. K. Ramakrishna Rao (Mysore); 15. Dr. Palaram.

Item 2: *MESSAGES OF INABILITY TO ATTEND THE MEETING.*

Messages of inability to attend the meeting received from the following members were read:—1. Dr. U. Krishna Rau (Madras); 2. Dr. G. V. Hanumantha Rao (Guntur); 3. Dr. B. P. Neogy (Calcutta); 4. Dr. J. Mojumdar (Calcutta); 5. Dr. P. Veeriah Chowdhary (Guntur); 6. Dr. T. V. Sivanandam (Coimbatore); 7. Dr. H. R. Dawar (Delhi); 8. Dr. T. N. Banerjee (Patna); 9. Dr. S. N. Saxena (Kanpur); 10. Dr. H. Hukku (Lucknow); 11. Dr. C. P. Bhat (Bhavnagar); 12. Dr. M. D. Patel (Ahmedabad); 13. Dr. K. Viswanadha Rao (Visakphapatnam); 14. Dr. M. N. Mahadevan (Bangalore); 15. Dr. D. K. Kothare (Bhuj)—Co-opted.

The following resolution was passed unanimously:

"The Working Committee of the Indian Medical Association notes with satisfaction that many Medical Men have been elected and nominated to the Central and State Legislatures, and while congratulating them, hopes that they will take active interest in all matters affecting Public Health and Medical care, consult the Indian Medical Association and be guided by it in shaping the future policy of the country in matters concerning the health of the Nation."

The Working Committee further congratulates those active members of the Indian Medical Association, who have been appointed as Ministers in various Governments.

Item 3: *CONFIRMATION OF THE PROCEEDINGS OF THE LAST MEETING OF THE WORKING COMMITTEE HELD AT AHMEDABAD ON THE 13TH AND 14TH FEBRUARY 1952.* (Circulated to the members as published in May 1952 issue of the Journal).

The Proceedings of this meeting had already been approved at the Annual Meeting of the Central Council held at Ahmedabad. These were now formally adopted.

Item 4: *BUSINESS ARISING OUT OF THE PROCEEDINGS OF THE LAST MEETING.*

(1) Pay, Status and Prospects of the Railway and other Medical officers.

The memorandum as approved by the Central Council has been sent to all the Governments and all branches of the I.M.A. This was noted and approved.

(2) Essential Drugs, Surgical Instruments and appliances for free import.

The House decided that since the Government had now further liberalised the import of these articles under the O.G.L. or by liberally issuing import licenses, this matter could be dealt with by the Central office and to bring it to the notice of the Working Committee only if found necessary.

(3) Fee Scales for Examinations of Life Insurance Cases.

Dr. Chamanlal M. Mehta informed the House that very recently he had had further discussion on the matter with the Secretary of the Insurance Companies' Association and he hoped that further progress may be expected.

(4) Mutual affiliation of the I.M.A. with the B.M.A.

(a) *Assets of the B.M.A. Branches in India*: According to the terms the British Medical Association had started to take steps for transferring the assets of its branches (to be dissolved) in India to the respective Provincial Branches of the Indian Medical Association. The process, however, would take sometime, said the Honorary General Secretary while he read out the relevant correspondence.

The Working Committee resolved 'that the Provincial Branches be requested to donate the amounts they might receive from the British Medical Association Branches to the I.M.A. "Building Fund."

(b) *Consideration of applications from branches for free copies* (50 *in number of the British Medical Journal*). The Committee decided that:

(1) Each Provincial Branch office should get one copy.

(2) The remaining copies to be distributed by the Honorary General Secretary according to his discretion.

(3) Out of the six other copies of the British Medical Journal for the Central Office, two were to be ear-marked for the Journal office, two copies to be given to the pool for distribution to the branches, one copy for office use and the remaining one copy to be kept for binding for record at the Central office.

(5) *Position regarding residencies and internships abroad.*

The Honorary General Secretary gave the following latest information to the members since the Selection Committee had met at Ahmedabad for the interviewing candidates:

Applications for residency appointments recommended by the I.M.A. to various hospitals in U.S.A. and Canada	65	
Number of candidates who have since proceeded to join their respective duties	36	No. of posts materialized 49
No. of candidates who have received the appointment letters but are due to proceed in the near future	6	
No. of candidates who refused to proceed after appointments had been obtained	7	
No. of candidates whose posts did not materialize or whose cases are under consideration	16	

The Honorary General Secretary placed on the table a scheme he had prepared for the selection of candidates in future. (*vide* Appendix "A").

Capt. Shivapuri while agreeing about the necessity of such a Scheme which was an improvement on the procedure followed thus far, requested the Honorary General Secretary to send full details to the Provincial Branches, so that the candidates may become conversant with the rules while applying.

General discussion followed, the main suggestions being that in the future selection of candidates, preference should not be given to candidates who possessed post-graduate qualifications or who had already held or were holding senior hospital appointments.

The House approved of the Scheme proposed by the Honorary General Secretary.

Dr. S. C. Sen, the Honorary General Secretary, then proceeded to read a letter he had written to the Medical Council of India re. recognition of certificates issued by the American hospitals and the Speciality Boards of the American Medical Association. The matter, he informed the house, was being pursued further.

Dr. Chamanlal M. Mehta suggested that the Universities in India be requested to recognise work done by Indian Post-graduate students in the U.S.A. or Canada, for Post Graduate Degree or Diploma examinations.

The Honorary General Secretary then read out a letter he had written to the United States Ambassador in India re. possibilities of further facilities for Post-graduate studies and training in American Hospitals. This was for the information of the House and he said future developments in this line would be reported in due course. Some general discussion followed in which the following members took part:—

Drs. B. R. Nayen, A. K. Bose & Ram Rakha Dheer.

It was now 5-25 P.M. and Dr. T. S. Tirumurthi, the President arrived. He occupied the chair after thanking Dr. Chamanlal M. Mehta for conducting the business of the house in his absence. Other members who had been held up at Khandwa also arrived.

(6) *System of appointment of Honorary Medical officers.*

The House directed the Honorary General Secretary to pass on the information available up-to-date to Dr. Chamanlal M. Mehta who was requested to prepare a draft for the consideration of the Joint Educational Scientific and Health Services Committee. The final report of the Committee was then to be circulated to the members of the Working Committee. Then afterwards the matter was to be taken up at the next meeting, if possible.

(7) *Consideration of a letter dated 17-7-51 of Dr. B. V. Mulay regarding a resolution passed by the Health Ministers Conference on the training of village Vaidyas.*

The Scheme now prepared by Dr. B. V. Mulay at the request of the Working Committee had been received. The Central office was directed to circulate it to the Provincial Branches for opinion.

(8) *Consideration of letter No. D/22/DHN/51 dated 8-8-51 from the Dhanbad Branch and letter No. 1290 dated 9-8-51 of Dr. B. V. Mulay, Sholapur, regarding some Indian Railways refusing to accept medical certificates issued by private medical practitioners to Railway employees.*

The relevant replies received from the Railway authorities were read out. The members wanted to know on what grounds was the certificate under question not accepted by the authorities; further enquiries were to be made on this point. It was decided to forward copies of all relevant papers to Dr. Mulay who was requested to make further enquiries at his end, where his certificate was not accepted by the Railway authorities in that area.

The case referred to by Capt. Shivapuri from the U. P. Provincial Branch was also to be further pursued.

At this stage there was some general discussion on the subject of medical certificates. Drs. C. S. Thakkar, B. C. Bose, T. S. Tirumurthi, B. V. Mulay, S. C. Sen and P. K. Guha took part in the discussion.

(9) *Consideration of Government of India Planning Commission Report—Five Year Plan especially Chapter 15 to 20 on Health, Housing, Education and Social Welfare.*

The Conveners of the five Regional Committees on the 'First Five Year Plan' were authorised to draft the Report and Dr. P. K. Guha was directed to put the recommendations in final shape and to send it on to the Honorary General Secretary. This report was to be sent to the Planning Commission and copies were to be circulated to all the members of the Working Committee and the Provincial Branches.

(10) *Relaxation of Rules re. Import Duty, Sales Tax and Excise Duty on essential medicines and appliances, drugs and medical books (Bengal Provincial Branch letter No. 580/50 51 dated 19-9-51).*

Action taken by the Honorary General Secretary was noted. This matter was not to be brought up again unless the Honorary General Secretary found it necessary.

(11) *Consideration of letter No. 63-51-52 dated 5-11-52 received from the Honorary Secretary, Bengal Provincial Branch re. conditions in Railway Medical Services and his suggestions and letter dated 13-11-51 of Dr. S. C. Seal of E. I. Rly. Hospital, Asansol, on the subject.*

Action taken by the Central office was noted. No further action was necessary as the recommendations of the Indian Medical Association on the subject of Pay, Status etc., of medical personnel also cover the sphere of Railway Medical Service.

(12) *Consideration of letter dated 19-10-51 of the W.M.A., New York, regarding holding of an International Conference on Medical Education.*

It was noted.

(13) *Plot of land for the Central office Building in Delhi.*

The Honorary Joint Secretary reported the progress achieved so far. The members felt satisfied that a suitable plot of land might be available in the near future.

(14) *Letter from Sitarampur Branch re. pay, status etc., of colliery medical officers.*

Noted. No further action was necessary. Only a copy of the recommendations for Pay, Status etc., is to be sent to the branch.

(15) *Consideration of letter No. 72 dated 8-1-52 of the Indian Chemical Manufacturers' Association and Proceedings of the Joint Meeting with the Working Committee of the I.M.A.*

It was decided to nominate four members at Calcutta to represent the Indian Medical Association on the Joint Committee of the I.M.A. and the Indian Chemical Manufacturers' Association.

The following were duly nominated as members of the Joint Committee:—1. Dr. A. K. Sen, (Calcutta); 2. Dr. A. C. Ukil (Calcutta); 3. Dr. A. D. Mukharji, (Calcutta); 4. Dr. R. Sinha, (Calcutta).

Item 5: (a) *FORMATION OF BRANCHES.*

The formation of following new branches were approved:

	Name of Local Branch	*Provincial Branch*	*No. of members*	*Date of formation*
1.	Rohtak ...	Punjab ...	21	1-10-51
2.	Qadian ...	do. ...	6	1-10-51
3.	Kot Kapura ...	do. ...	5	1- 4-52
4.	Faridkot ...	do. ...	7	1- 4-52
5.	Bhusaval ...	M. and Karnatak	13	1- 4-52
6.	Vengurla ...	do. ...	8	1- 4-52
7.	Sangamner ...	do. ...	7	1- 4-52
8.	Pachora ...	do. ...	5	1- 4-52
9.	Kothagudium ...	Hyderabad-Dn.	11	1- 4-52
10.	Khandwa ...	Madhya Pradesh	13	1- 4-52
11.	Kurasia Coal Field ...	do. ...	7	1- 4-52
12.	Umreth ...	Gujarat and Saurashtra ...	6	1- 4-52
13.	Anamalais ...	South India ...	6	1- 4-52
14.	Choharpur ...	Uttar Pradesh ...	6	1- 4-52
15.	Dantan ...	Bengal ...	7	1- 4-52
16.	Kharagpur ...	do. ...	23	1- 4-52
17.	Cooch Behar ...	do. ...	15	1- 4-52
18.	Ramjibonpur ...	do. ...	9	1- 4-52
19.	Bolpur-Viswabharati ...	do. ...	11	1- 4-52
20.	Kolar Gold Field	Mysore ...	9	1- 4-52
21.	Chichmagalpur	do. ...	9	1- 4-52

(b) *REVIVAL OF BRANCHES.*

The following branches were revived:—

1.	Puinan ...	Bengal ...	12	1-10-52
2.	Jaunpur ...	U.P.	6	1-10-52

(c) *SUSPENSION OF BRANCHES.*

1.	Nagapatam ...	S. India ...	1- 4-52
2.	Dumka ...	Bihar ...	1-10-51
3.	Agra ...	U.P.	1- 4-52
4.	Chandausi ...	do.	1- 4-52
5.	Mainpuri ...	do.	1- 4-52
6.	Oral ...	do.	1- 4-52
7.	Pilibhit ...	do.	1- 4-52

Item 6: *CONSIDERATION OF C.F.C. ARREARS.*

After due consideration, the following outstanding arrears were written off.

	Name of the Provincial Branch	*Amounts to be written off* 1948-49 Rs.	1949-50 Rs.	1950-51 Rs.	1951-52 Rs.
1.	M. and Karnatak ...	58/12/-	...	...	...
2.	Rajputana ...	92/12/-	...	...	...
3.	Andhra ...	...	260/-	...	...
4.	South India ...	...	...	...	95/-
5.	Assam ...	...	...	...	4/-

The Honorary General Secretary gave the latest position re. C.F.C. arrears and realisations up-to-date.

On the recommendation of the Honorary General Secretary, it was decided to write off the outstanding amount of Rs. 85-15-6 against Shree Gurudas Bandopadhaya an ex-employee of the I.M.A. Central office, as advised Mr. R. K. Deb, Honorary Legal Adviser, I.M.A., Calcutta, in his letter No. R/196 dated 18th June, 1952.

Item 7: *ADOPTION OF AUDITED ACCOUNTS.*

(1) Central office (from 1-10-51 to 31-3-52)
(2) Journal Dept. (do. do.)

The Audited Accounts of the Central office and the Journal office for the quarter ended 31-3-52—as circulated with the Agenda, were duly adopted.

The Honorary General Secretary was authorized to open another current account in any other suitable bank instead of keeping the whole amount of the current Central Funds in one bank.

The following resolution was passed:—

"Resolved that a Current Account (Central Fund) or Short Term Fixed Deposit account in the name of the Indian Medical Association be opened with the Allahabad Bank Ltd., Delhi. This account as and when opened, would be operated jointly by either Dr. S. C. Sen, Honorary General Secretary, or Dr. A. P. Mittra, Honorary Joint Secretary, and Dr. H. R. Dawar, the Honorary Treasurer."

At this stage at the request of the Bihar members, the provisional dates of the next Conference at Patna were approved:

Working Committee:—21st and 22nd December, 1952
Annual Central Council meeting:—23rd and 24th Dec., 1952.
No engagement but the Central Council might use this day, if necessary—25th December, 1952.
Open Session of the Conference:—26th, 27th and 28th December, 1952.

Dr. S. C. Sen, the Honorary General Secretary, informed the members that Dr. Louis H. Bauer, President-Elect of the American Medical Association, Dr. D. Knutson, President of the World Medical Association and Dr. Morris Fishbein were likely to visit India for a week or ten days in October. There would be no expenses to be incurred by the Indian Medical Association for their travelling or stay. A programme was to be prepared by the Honorary General Secretary for these distinguished doctors so that they could meet the members of the Indian Medical Association and address them at several centres.

The President announced that the next meeting of the Working Committee would be held at Vishakhapatnam on 25th and 26th October, 1952.

Item 8: *CONSIDERATION OF RESOLUTIONS OF THE UTTAR PRADESH, KANPUR AND CALCUTTA BRANCHES REGARDING BACTERIOLOGICAL (BACTERIAL) WARFARE IN KOREA.*

The Honorary General Secretary read out the relevant correspondence and the resolutions which had been received from the Lucknow, Kanpur and Calcutta Branches and the Executive Committee of the U. P. State Branch. He had been asked by these branches to forward these resolutions to

the Prime Minister and to the Secretary-General of the U.N.O. He asked the House whether he had to forward all resolutions thus received or he had any discretion in the matter. In case of these resolutions under consideration, he preferred to bring them to the notice of the Working Committee instead of forwarding them as desired, as the Central Council at Ahmedabad in February, 1952, had decided that the Indian Medical Association, as a body, should not take part in any International political question particularly when it was of a controversial nature, *viz.*, the question of alleged use of Bacteriological Weapons in the Korean War. Similar was the position of the Indian Medical Association re. an International Peace Congress of Doctors which was proposed to be held in Rome.

At this stage, one member (Dr. A. K. Bose) observed that the resolutions were not political in nature but the Honorary General Secretary was dragging politics into them. The Honorary General Secretary again read out the resolutions and pointed out that in them all names of political personalities and names of the belligerent countries and organizations had been mentioned and thus these resolutions at once became political in nature.

Dr. A. C. Ukil submitted that in his opinion, the Indian Medical Association was quite competent to pass a resolution on the subject on humanitarian grounds but no mention should be made of Korea or any other country, otherwise it would become political in nature.

Much discussion followed in which practically all the members present took part.

The President, Dr. T. S. Tirumurthi said that the Honorary General Secretary had discretionary power to withhold any resolution or letter which he might be asked by a branch or branches to forward elsewhere and he could bring the matter to the notice of the Working Committee for obtaining its directions. The President then put the question whether the Working Committee would like to pass a resolution on the subject of Bacterial Warfare on general humanitarian principles or not. He asked for votes. The result of the voting was that 12 members were in favour of passing such a resolution and 9 were against it. The decision of the House by majority was that a resolution on general lines should be passed and the Honorary General Secretary was authorised to draft the same and give it publicity. Since then, the Honorary General Secretary has drafted the following resolution:

"The Working Committee of the Indian Medical Association resolved that all Nations should agree, on humanitarian grounds, to ban Bacteriological warfare."

Item 9: *CONSIDERATION OF PROPAGANDA GRANTS.*

(1) Letter No. 580/51-52 dated 1-5-52 of Bengal Provincial Branch for a grant of Rs. 500/-.

(2) Letter No. 750/50-51 dated 7-8-51 and No. 800/50-51 dated 10-9-51 of the Punjab State Branch for a grant of Rs. 500/-.

(3) Letter No. 604/51-52 dated 17-5-52 of the S. Indian Provincial Branch re. tour of a member for membership drive from a different Provincial Area etc., and grant from the Central Funds.

The Honorary General Secretary read out the above letters. He also read out the Circular No. 18 dated 18th March 1952 which was based on the opinion of the Working Committee taken when the budget for the current year was made.

After some discussion the House decided not to approve the grants applied for in the above mentioned three cases.

Item 10: *LETTER DATED 10-5-52 OF DR. CHAMANLAL M. MEHTA, BOMBAY, RE. MILITARY TRAINING TO ALL MEDICAL GRADUATES AND COMPULSORY POST-MORTEM EXAMINATION WHEN DIAGNOSIS IS DOUBTFUL.*

The Honorary General Secretary read out the relevant letter after which there was some discussion on the subject. The House felt that Government at the present stage were not likely to agree to introduce compulsory Military Training for all medical graduates due to financial implications.

The House decided to request the Central and State Governments to pass necessary legislation for compulsory post-mortem examinations in all cases of doubtful diagnosis. The relevant resolution of the All-India Medical Conference on the subject might again be forwarded to the authorities and their attention drawn to it.

The Provincial Branches were also to be requested to move their respective State Governments on the subject of post-mortem examinations.

Item 11: *LETTER NO. F. 3G 51/377 DATED 13-5-52 OF THE MEDICAL COUNCIL OF INDIA RE. POST-GRADUATE DIPLOMAS AND ELIGIBLE QUALIFICATIONS FOR TEACHERSHIP IN A MEDICAL COLLEGE AFFILIATED TO A UNIVERSITY.*

The Honorary General Secretary read out the letter under consideration for the information of the House on the subject of recognition of Post Graduate certificates granted by American Speciality Boards and recognised U. S. Hospitals by the Medical Council of India and the Indian Universities.

The action taken by the Honorary General Secretary was approved and noted.

Item 12: *LETTER NO. 1165/53/51-52 DATED 8-4-52 OF CAPT. H. N. SHIVAPURI, HONORARY SECRETARY, U. P. STATE BRANCH REGARDING DR. O. P. GUPTA, M.B. (NATIONAL) AND MEMBERSHIP OF THE INDIAN MEDICAL ASSOCIATION.*

After going through all the details of the case, it was decided that (Dr.) O. P. Gupta could not be enrolled as a member of the Indian Medical Association since he had not been registered by any State Medical Council.

Item 13: *LETTER NO. F. 25/4/51-MI DATED 19-4-52, FROM THE MINISTRY OF HEALTH, GOVERNMENT OF INDIA, CONTAINING OBSERVATIONS OF THE GOVERNMENT OF INDIA, ON RESOLUTIONS OF XXVII ALL-INDIA MEDICAL CONFERENCE HELD AT SHOLAPUR.*

The replies received were noted.

Item 14: *LETTER OF HONORARY JOINT SECRETARY, INDIAN MEDICAL ASSOCIATION (Central office) ADDRESSED TO HON'BLE RAJKUMARI AMRIT KAUR, MINISTER OF HEALTH, GOVERNMENT OF INDIA RE. PROGRESS AND PLANNING FOR THE FUTURE PERTAINING TO EXPANSION OF MEDICAL SERVICES, MEDICAL TRAINING AND ESTABLISHMENT OF MEDICAL INSTITUTIONS WITH SPECIAL REFERENCE TO ALL-INDIA MEDICAL INSTITUTE AND ACTIVITIES OF THE W. H. O. IN THIS COUNTRY.*

Dr. A. P. Mittra, the Honorary Joint Secretary read out the letter which had been written from the Central office and the reply received from the Ministry of Health. He also read out the letter written by the Honorary General Secretary to the Union Health Minister.

The action taken by the Honorary General Secretary was approved. Further developments on the subject were to be reported to the Working Committee in due course.

Item 15: *LETTER DATED 12-4-52 OF DR. S. C. SEAL, M.B., B.S., E. I. R. HOSPITAL, MADHUPUR S. P. SUGGESTING FOR ESTABLISHING RAILWAY BRANCHES.*

The House did not approve of the Scheme suggested by Dr. S. C. Seal of Madhupur re. formation of special Railway Branches of the Indian Medical Association.

Item 16: *LETTER NO. 503/51-52 DATED 1-4-52 RECEIVED FROM THE BENGAL PROVINCIAL BRANCH TO ARRANGE FOR A DETAILED RECORDING OF THE PROCEEDINGS OF THE WORKING COMMITTEE AND THE CENTRAL COUNCIL AND CIRCULATION OF THE SAME TO THE PROVINCIAL BRANCHES WITHIN A REASONABLE TIME.*

The letter was considered. The House decided that it was not possible to have full details of the proceedings of Working Committee meetings unless several Stenographers

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were employed on the shift basis and this involved much expense. Attempts were always made by the Secretaries to give a factual report with essential details and this effort would be maintained with an eye on further improvement as far as possible.

Item 17: *LETTER NO. F. 11-11/52/52D.S. DATED 22-4-52 OF THE UNDER SECRETARY, GOVERNMENT OF INDIA, MINISTRY OF HEALTH RE. DRUGS AND MEDICINES MANUFACTURED BY INDIAN FIRMS—NEED FOR POPULARISATION OF —RECEIVED WITH A LIST OF FOREIGN PRODUCTS WITH INDIAN SUBSTITUTES AND LETTER DATED 27-5-52 OF DR. A. K. SEN, VICE-PRESIDENT, I.M.A.*

The letter was considered and the action of the Central office in referring the matter to Dr. A. K. Sen, Senior Vice-President (the Representative of the I.M.A. on the Drugs Advisory Board) was approved. Dr. A. K. Sen informed the House that he was in correspondence with the Pharmaceutical Manufacturer's Association and that he would report further developments in due course.

Dr. S. C. Sen suggested that the Central office be permitted to enquire from Government on what basis they were pleased to recommend the preparations of Indian Origin as substitutes of reputed foreign preparations and especially if these recommended preparations had been analysed and certified to be correct according to the claims of the respective manufacturers.

The House approved of the action proposed by the Honorary General Secretary.

Item 18: *LETTER NO. 561(3)/51-52 DATED 24-4-52 AND NO. 639(3)/51-52 DATED 24-5-52 OF THE BENGAL PROVINCIAL BRANCH, I.M.A. TO DR. T. N BANERJEA, IMMEDIATE PAST PRESIDENT RE. ITEM NO. 18 OF THE PROCEEDINGS OF THE WORKING COMMITTEE HELD ON 13TH AND 14TH FEBRUARY 1952 AT AHMEDABAD.*

The letter under question was read re. the request to delete a certain line of the Proceedings of the Working Committee held on 13th and 14th February 1952 at Ahmedabad. The Honorary Joint Secretary, Dr. A. P. Mittra also read out a letter by Dr. T. N. Banerjea requesting the deletion of the passage and that he would have corrected the Proceedings had he been shown these before publication in the Journal.

Dr. A. P. Mittra submitted to the House that Dr. Banerjea was not correct in saying that he had not seen the Proceedings. Dr. Banerjea had probably forgotten that the proceedings in question after being written, and cyclostyled, were later signed by himself after the conclusion of the working committee meeting on 14th February, as these had to be presented the next day, i.e., the 15th February 1952 at the Annual Meeting of the Central Council. The Proceedings were read and approved of at the said Central Council meeting, when no objections were put forward and Dr. Banerjea himself was presiding.

Dr. Mittra felt that these Proceedings had been approved of by the Central Council—the Supreme authority of the Indian Medical Association. If any deletions or additions had to be made, only the Central Council could do it at its next meeting.

The House referred the matter to the Central Council for consideration at its next meeting.

Item 19: *LETTER NO. 57 DATED 30-5-52 OF THE HONORARY SECRETARY, JAMSHEDPUR BRANCH, REGARDING REFUND AGAINST THREE RAILWAY TICKETS PURCHASED FOR MADRAS DOCTORS ATTENDING THE WORKING COMMITTEE MEETING AND CREDIT OF RS. 73/8/- FROM THE CENTRAL FUND PAID LESS BY THE S. INDIAN PROVINCIAL BRANCH.*

The whole matter was fully discussed and those members of the S. Indian Provincial Branch who were present at the meeting, explained their viewpoint. The House decided that the request of the Jamshedpur Branch for adjusting the amount of Rs. 73/8/- against its C.F.C. at the Central office could not be entertained. The House also felt that Jamshedpur as a branch should not suffer this loss and that the Honorary General Secretary should write to Dr. P. A. S. Raghavan and request him on behalf of the Working Committee to pay up this small amount to the Honorary Secretary, Jamshedpur Branch, I.M.A. Jamshedpur.

Item 20: *LETTER NO. 31 DATED 31-5-52 REGARDING REGISTRATION OF I.M.A. BRANCHES (UNDER MAHARAHSTRA AND KARNATAK) UNDER THE PUBLIC TRUST REGISTRATION ACT BOMBAY.*

After the relevant correspondence had been read, it was decided to seek the advice of the Honorary Legal Adviser on the matter. It was also decided to get legal opinion at Bombay through the Bombay Provincial Branch for clarification of the point.

Item 21: *LETTER DATED 31-5-52 OF DR. T. S. TIRUMURTI, PRESIDENT, CONTAINING A RESOLUTION RECOMMENDING THE PUBLICATION OF I.M.A. BOOK OF MEDICAL SCHOLARSHIPS IN THE UNION OF INDIA ON THE BASIS OF THE B.M.A. BOOK OF MEDICAL SCHOLARSHIPS.*

Dr. Tirumurti withdrew the proposal.

Item 22: *ANY OTHER BUSINESS WITH THE PERMISSION OF THE PRESIDENT.*

1. Letter No. 201/51-52 dated 16th June 1952 of the South Kanara Branch (under South Indian Provincial Branch) re. publication and printing the names of all the members of the Association to facilitate nominations and elections of President and Vice-Presidents.

The House considered the letter, but was of the opinion that at present it was not possible to accept the suggestion.

2. Letter No. 607/51-52 dated 17th May 1952 of the Hony. Secretary, South Indian Provincial Branch, I.M.A., Coimbatore, to Registrar, Madras University, re. concession to medical licentiates for obtaining M.B. B.S. Degree.

As the matter referred to in this letter was a matter for the Madras University, the Working Committee decided that no further action was necessary on the part of the Central office of the Indian Medical Association.

3. Letter No. 644/51-52 dated 3rd June, 1952 of the Honorary Secretary S. Indian Provincial Branch, I.M.A., Coimbatore, re. formation of more branches in the city of Madras and letters dated 17th and 25th June 1952 of Madras Branch in this connection.

It was pointed out that if complete proposals were made, the Working Committee (and the Central Council) might probably approve of the formation of several branches in the city of Madras. It was at the same time pointed out that it would not probably be a wise move as none of the branches would be in a solvent position and be able to carry on the work efficiently. It was further pointed out that there would not be any possibility of the Madras city being given the Provincial status according to the present rules of the I.M.A.

ACKNOWLEDGMENTS: The following two resolutions were moved from the chair and passed unanimously:—

(1) This meeting of the Working Committee of the Indian Medical Association places on record its deep appreciation and thanks for the hospitality and courtesy accorded to its members on the occasion of the meeting held at Indore on 28th and 29th June, 1952, by the Indore Branch and the Madhya Bharat Provincial Branch of the I.M.A.

2. The Working Committee of the Indian Medical Association offers its grateful thanks to the authorities of the M.G.M. Medical College, Indore, for permission to hold the meeting of the Working Committee in the Tagore Library Hall of the college at Indore.

With a vote of thanks to the chair, the meeting terminated.

Sd./- S. C. SEN,
Honorary General Secretary.

Sd./- T. S. TIRUMURTI,
President

APPENDIX 'A'

INDIAN MEDICAL ASSOCIATION

(Central office)

Instructions for Selection of Candidates for Internships and Residencies Abroad

1. Only members of the Indian Medical Association who have good knowledge of the Indian Medical Association, its principles and workings, will be accepted.

2. Ordinarily only young doctors—recently qualified and willing to stay abroad for 3-4 years should be selected.

In U.S.A. and Canada, no diplomas like D.O.M.S., D.M.R.E., M.R.C.P., F.R.C.S. etc., are issued. As such, doctors going to U.S.A. and Canada for training in specialities will not be able to add letters after their names. There are, however, about 14 specialities for which doctors can get trained. After training (which usually last for 3—4 years) in approved hospitals, candidates can sit for Diploma examination of Speciality Boards. If successful, candidates will get Diplomas which they can frame and hang in their consultation rooms. These Diplomas are highly thought of in U.S.A. and Canada and are accepted as evidences for senior hospital and teaching appointments.

The Medical Council of India is being approached and it is hoped that it will recognize these diplomas for teaching and hospital appointments. In addition, it may be possible, in some cases, where the hospital is attached to a University, to work for M.Sc. or Ph.D. degrees.

3. Fee of Rs. 5/- with each application is to be sent to the Central office by the Provincial Branches. This is not refundable and is to be credited to the Central office of the Indian Medical Association.

4. Candidates selected finally by the Central Selection Committee must deposit at least Rs. 500/- (not refundable) before their names are sent up to the hospitals abroad. This will indicate their bonafide intentions and will be used for deposit against passage.

5. Applications from men in services will only be considered if the application is supported by proof that there will be no difficulty about leave of absence and release, whenever the appointment is likely to mature.

6. Provincial and Local Branches must make sure that candidates selected by them have necessary funds (about Rs. 10,000/-) at their disposal, and that there will be no difficulty about health certificates, passports and visas, and about their departure, *even at short notice.*

7. Only those used to Western methods of living and willing to have usual Western type of food, should apply and be selected.

8. Candidates must possess good command of spoken and written English. They must not have accents not understood by Americans.

9. Applications should be submitted as per preforma enclosed.

S. C. Sen,
Honorary General Secretary,
I.M.A., Central office.

INDIAN MEDICAL ASSOCIATION
(Central office)

Application as per proforma to be submitted in quadruplicate —3 copies to be sent to the Central office, along with a fee of Rs. 5/-

PROFORMA:

Subject for Study

1. Name———————————————
2. Address———————————————
3. Married or Single———————————————
4. Member of———Branch. (————Provincial Branch)
   Date of Joining I.M.A.————————————
5. Qualifications
   (*a*) Academic———————————————
   (*b*) Experience———————————————
   (*c*) Other qualifications———————————————
6. Copies of Testimonials, not more than three, to be attached—one of which must be from the Dean or Principal of the Medical College———————————
7. Names and addresses of two references to be sent.
   1. ———————————————
   2. ———————————————
8. THREE copies of Passport size photograph—with name and addresses in Block letters—to be attached with the application.

TRAVANCORE-COCHIN MEDICAL CONFERENCE

The second annual Conference of the Travancore-Cochin Medical Association was held at the Government House, Kottayam on the 24th November, 1951. One hundred and thirtynine delegates from different branches were present. The general session was preceeded by a business meeting in which Dr. N. Krishnan Tampi presided. After the speech of Shri G. Chandrasekhara Pillai, the Minister for Health of the Travancore-Cochin State, Dr. Kesavan Nair, the hony. secretary presented the annual report of the branch for the year 1950-51. The report showed that the membership of the Association had risen from 344 to 441. It also contained a brief account of the activities of the branches. The statement of accounts was also presented and passed. New office-bearers were then elected with Dr. C. O. Karunakaran as president and Dr. R. Kesavan Nair as secretary for the year 1951-52.

The general session commenced at 2-30 p.m. The Chairman of the Reception Committee Dr. P. T. Thomas welcomed the delegates and the distinguished visitors in a neat little speech. Next Dr. P. V. Cherion inaugurated the Conference. He also presided over its deliberations. Dr. Cherion in course of his address, said,

"With the progress of knowledge, prevention of diseases has become more important than cure. Proper maintenance of health of the whole population should be our aim, and for this more medical men are required. Unfortunately the present number is insufficient. In Russia they started the Felcher system, training people for shorter period, to overcome this shortage. We cannot accept this. They have themselves given it up. A proper knowledge of scientific medicine cannot be had with a short period of medical education, and the standard should never be lowered.

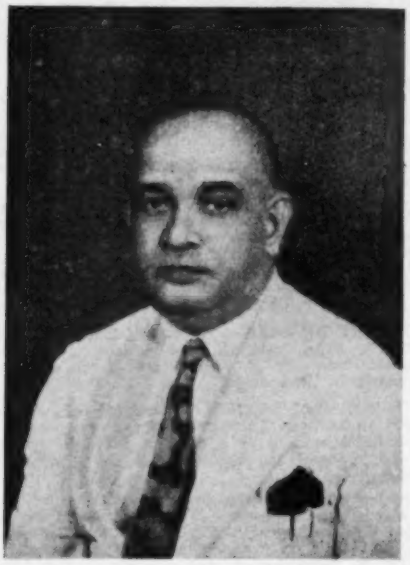
The All-India Medical Council has suggested a degree course of four and a half years, with one year of house surgeoncy. No person should be allowed to practice without the practical training derived from the house surgeoncy. In certain colleges shift system was introduced to train more students. Medical education needs more of individual attention. In olden days the number of students was not large and the teacher knew both the strength and the weakness of his pupils. But now the number has become so large that no personal contact is possible. It would be better to close down an institution rather than admit such large numbers and train them by the shift system.

Referring to the indigenous systems of medicine he continued, "Unfortunately we who have been trained scientifically cannot accept these systems though we are asked to accept them. Ayurveda and Unani are systems of arrested growth. They stopped growing a few hundred years ago, probably due to suppression by outside agencies. My advice would be that you should not condemn the other systems of medicine. I have seen cases cured by other systems where we have failed. But I am against the training in indigenous medicines up to the University standard, though I agree with part of their training.

Medical relief can be cheaper only if medicines become cheaper. Proper treatment can be given only if genuine drugs are available, and India has to depend on other countries for them. If the Government allow duty free import

of drugs treatment can be made much cheaper and the common man will benefit.

A word about the medium of instruction. There is a cry for teaching even scientific subjects in Hindi. In my opinion English should continue as the language in which scientific subjects are taught. It is spoken all over the world and it gives easy access to progress in every branch of science. Even European text books are translated into English. A false sense of patriotism should not make us sacrifice English. It will result in a lowering of standards and ultimately drag the country down.



DR. P. V. CHERIAN, THE CHAIRMAN OF THE CONFERENCE.

Education should not be interfered with by external influences. Universities should be autonomous. They should lay down standards regarding education and selection of teachers. Government at political influences are likely to reduce these standards and act detrimentally to the progress of education. There are certain services associated with medicine which deserve attention, particularly nursing. Nursing in India is a very neglected service. Before the attainment of Independence, this service was almost the monopoly of Anglo-Indians. Now the pay is poor and the work hard with little provision for other comforts, and so there was no impetus for the more respectable class of girls to take up this profession. Nursing is a profession that deserves generous support. Even humanitarian work should have its return in elementary comforts. We cannot expect the selfless devotion of Florence Nightingale or Roman Catholic nuns in all, and the status and pay of nurses should be improved.

There is much talk about medical men serving in the villages. Villages form the backbone of the country and the villagers' health must be looked after. But the medical profession should not be penalised by the imposition of compulsory work in rural centres. Are the conditions in the villages attractive for the medical men trained in the cities and used to certain amenities? Are there proper schools for their children or decent quarters for their living? Can any other highly educated professional man be asked to stay in the village and will he do it if asked? The only solution will be to bring the cities to the villages and the villages to the cities. Every village cannot be given well equipped hospitals or sanatoria, but it can be brought closer to them by improving communications. The question of tackling emergencies in the villages requires consideration.

Our profession owes a great duty to the public. Medical education is financed partly by the Government and partly by the guardians or parents of students. The Government puts into it more than the student contributes. Therefore one should take up the profession only after mature consideration. Many of us have become medical men accidentally and not after proper consideration. One must decide on his future profession as early as possible even at the school stage and prepare himself from that time for his professional education. There should be a proper background of the other basic sciences. Because of its importance, pre-registration course is coming back in many western countries. A proper sense of duty and responsibility is lacking in us. We do not put our heart and soul into our profession. Every human life is important. It may be that we had copied the idea from our foreign masters that poor human lives were not valuable. We should get rid of that idea and respect every human life.

The ancillary profession of dentistry has not developed sufficiently in this country though it is very popular and remunerative in the West. The National Health Scheme is commendable as it brings medical relief to every citizen of the State. Some similar scheme should be introduced in this country also. The children are the future citizens and their health is of great value. There should be proper medical inspection of school children and an efficient follow up. Then and only then can a healthy nation be built up. All these matters deserve our attention individually, and collectively through our Associations.

The session came to a close at 4 p.m. and the scientific session commenced its sitting at 5 p.m. with Dr. P. V. Cherion in the chair. The following papers were read:—

(1) Coronary Diseases—by Dr. A. K. Menon.
(2) Cancer Cervix—diagnosis and treatment—by Dr. P. Kalyanikutty.
(3) Some Public Health Problems—by Dr. R. M. Mathew.

The following resolutions were adopted in the Conference:—

1. This Association places on record its deep sense of sorrow at the demise of Dr. (Mrs.) T. Francis, and Dr. M. K. Gopala Pillai, two of its active members.

2. Resolved to request the Indian Medical Association to move the Central Government to give preference, other conditions being equal, to children of doctors, in the matter of admission to the Medical Colleges.

3. The fee fixed by the Travancore-Cochin Government for the issue of vaccination and inoculation certificates in International forms is one rupee per head. It is degrading for a medical officer to accept this paltry sum. Resolved that to keep up the dignity of the profession the certificate should be given free if the fees cannot be raised.

4. This Association requests the Government and the University not to include any modern medical subjects in the curriculum of the Ayurvedic courses as the practice of modern medical subjects with incomplete knowledge will be dangerous to the public.

5. This Association views with concern the Order passed by the Travancore-Cochin Government regarding the re-entertainment of retired medical officers, as their appointments to responsible places will hamper the chances of younger people in the department getting the experience obtainable from such posts.

6. This Association requests the Government of Travancore-Cochin to institute the honorary system as is in vogue in Madras, to overcome the paucity of hands. Appointment of any retired medical officer, who is to be re-entertained, should be as an honorary medical officer with a limited number of beds.

7. This Association requests the Travancore-Cochin Government to take early steps to implement the integration of medical services as the due promotions and increments of pay are withheld pending integration.

8. This Association requests the Travancore-Cochin Government to enforce the penal clause in the Medical Practitioners' Act at an early date to prevent abuse of medicines by unauthorised people with disastrous result.

9. Resolved that the rule pertaining to the number of Vice-Presidents be amended as follows:

"There shall be three Vice-Presidents."

BRANCH PROCEEDINGS

ASSAM PROVINCIAL BRANCH—A meeting of the branch was held on 20-7-52 with Dr. Lala S. S. Das in the chair. Seven members were present. A Managing Committee was formed, which would meet every month. The general body, it was decided, would meet once in 3 months.

BEZWADA BRANCH—The annual general meeting of the branch was held on 27-7-52 with Dr. K. Vasudeva Rao in the chair. A condolence resolution was passed on the death of Dr. A. Narasinham and Dr. Kameswaramma. After the normal business, new office-bearers were elected. The president elaborately explained the various aspects of Chemotherapy in Tuberculosis and also of B.C.G. Dr. P. Venkateswara Rao read a paper on Acute Appendicities with Special reference to the early diagnosis of atypical cases.

CACHAR BRANCH—A meeting of the branch was held on 26-7-52 with Dr. S. K. Datta in the chair. Sixteen doctors were present. After the normal business was over, Dr. M. K. Chaudhury read a paper on Hæmoptysis.

Another meeting of the branch was held on 28-6-52. Twentytwo members were present. Dr. S. K. Dutta was in the chair. Capt. L. Rahman read notes of a case of Laryngeal concer with metastatic glands in the neck.

COIMBATORE BRANCH—A meeting of the branch was held on 2-8-52 with Dr. I. Pitchari Robert in the chair. The bye-laws of the Association were adopted. A symposium on "Peptic Ulcer" was arranged in which Drs. Jayakumar, Subramaniam, Leelakrishnan and Warrior took part.

ERODE BRANCH—A meeting of the branch was held on 5-7-52 with Major R. V. N. Nayudu in the chair. Dr. V. Govindan Nair spoke on "Venereal Diseases, their clinical features and modern methods of treatment."

GIRIDIH BRANCH—A meeting of the branch was held on 22-6-52 with Dr. H. C. Chatterjee in the chair. Twelve members were present. The members offered congratulutions to the doctors who were elected to the State Legislatures and the Parliament. In the Essential Goods Bill which was introduced in the Parliament, the members opined that medicine and invalid foods should be included.

GHUGUDANGA BRANCH—A meeting of the branch was held on 27-7-52 with Dr. S. Sarkar in the chair. Seven members were present. Dr. S. C. Sen Gupta read a paper on "Intestinal Myiases."

KANPUR BRANCH—The branch celebrated the Independence Day at the Temple of Service on the 15th August. A clinical meeting of the branch was held on 17-8-52 with Dr. B. N. Bhallay in the chair. Dr. B. B. Bhatia, Professor of medicine, K. G. Medical College, Lucknow, delivered a lecture on "Trend of recent advances in modern medicine."

MADRAS BRANCH—The Madras branch of the I.M.A. arranged a tea party on 6-5-52 at the Madras Medical College to felicitate Dr. T. S. Tirumurti on his election as President of the I.M.A., Dr. U. Krishna Rao and Dr. M. V. Krishna Rau on their appointments as Ministers in the Madras cabinet. Dr. K. Narayanamurthi, president of the Madras branch was in the chair. It was a very pleasant function and many distinguished persons were present on the occasion.

MADURA BRANCH—A meeting of the branch was held on 19-7-52. Dr. M. N. Raian was in the chair. Dr. G. C. Anbunathan read an interesting paper on "Antibiotic Therapy in General Practice." Dr. J. J. Barnes, presented a radiograph of the Pelvis in a case of Carcinoma of the Prostrate with secondaries in the bony pelvis. Dr. H. S. Thomas gave a brilliant review of his tour of U.S.A. with colour slides.

MALABAR BRANCH—A meeting of the branch was held on 26-7-52 with Dr. C. K. Menon in the chair. Thirty members were present. The president declared open the Reading Room. Two interesting papers on (1) Shoulder Pain by Dr. C. K. Menon and (2) Prolonged Pyrexias by Dr. C. R. Parasuram were read.

MALEGAON BRANCH—A meeting of the branch was held on 20-7-52. All members were present. It was decided to start a medical library of the branch. Dr. G. H. Thuse spoke on "Addisons' disease."

MUZAFFARPUR BRANCH—A meeting of the branch was held on 27-7-52 with Dr. N. N. Gupta in the chair. Ten doctors were present. Capt. Das Gupta read a paper on "Ascitis."

SABARMATI BRANCH (SABARMATI MEDICAL SOCIETY I.M.A.)—A meeting of the branch was held on 19-7-52. Dr. Miss Tanumati Seth spoke on "Dysmenorrhœa in Practice."

In the monthly meeting of August 1952 Dr. H. J. Mehta read a paper on "Acute Dysentrics."

SHIMOGA BRANCH—A meeting of the branch was held on 16-8-52. Dr. M. V. Shanbhag was in the chair. The following subjects were discussed, (1) a. A cases of Unusual Menifestation of Neural Leprosy and b. Lepramatous Pyrexia by Dr. K. Mohideen Burahan (2) Case Reports of Hepatitis by Dr. S. Gurumurthy.

TANJORE BRANCH—A meeting of the branch was held on 15-6-52 with Dr. N. R. Subramaniam in the chair. Thirty two members were present. A condolence resolution on the death of Dr. U. Rama Rao was passed. Dr. P. V. Cherian, Dr. U. Krishna Rao, Dr. M. V. Krishna Rao and Dr. Nathamuni were felicitated on their assumption of offices in the Madras Legislative Assembly and Council.

Another meeting of the branch was held on 27-7-52 with Dr. N. R. Subramaniam, in the chair. Thirty Eight members were present. A resolution was passed authorising the president to take suitable steps for collection of funds to construct a building to house the association. Dr. P. Narayana Rao then demonstrated a case of Pruritis. Dr. K. Ramchandran spoke on "Modern Trends in the treatment of peptic ulcers.

TIRUCHY BRANCH—A meeting of the branch was held on 12-7-52. Fifty members were present. Dr. Srinivasan was in the chair. Dr. P. Natesan gave a talk on "Recent developments in Venereology."

TIRUNELVELI BRANCH—A meeting of the branch was held on 28-6-52. Thirtysix members were present. Dr. S. Chandrasekharam gave a talk on "Pleural Effusions." Films on "Tolserol in Rheumatism" and "Anæsthesia" were also shown.

Another meeting of the branch was held on 26-7-52. Fifty members were present. Ten distinguished guests also attended the meeting. Dr K. Chintan Nambiar gave an interesting lecture on "Recent Advances in Neurosurgery." Next Dr. R. Subramaniam read a paper on the "Management of Hypertension."

TRAVANCORE-COCHIN STATE BRANCH—A special meeting of the branch was held in the Auditorium of the Medical College, Trivandrum on 7-1-52, in honour of the visit of the Hon'ble Sri Rajkumari Amrit Kaur. About 120 members from the branches were present. The president of the association, Dr. C. O. Karunakaran in welcoming the Hon'ble Minister, invited her attention to the following problems, (1) the tendency to lower the standard of medical education by introducing shorter courses, (2) dilution of modern medicine with indigenous medicine (3) shortage of essential drugs and instruments (4) flooding of the market by spurious or adulterated drugs and chemicals (5) need for immediate enforcement of food and drug act. The Hon'ble Minister in her reply dwelt at length on all the problems mentioned by the president of the association and said, "I can assure you on behalf of the Government of India that you will certainly have our co-operation in anything which you wish to do for the betterment of the practice of your profession."

UTTAR PRADESH STATE BRANCH—The third meeting of the working committee of the Branch Council was held on 22-6-52. Four members were present. At the outset a condolence resolution was passed on the death of Dr. V. P. Gupta of Meerut. Re. the letter of the D.M.H.S., U.P. regarding the reporting of sudden unnatural deaths to proper authorities, the committee made certain suggestions and declared that the association would oppose if the matter is placed in the hands of the police at any stage. Regarding Dr. Atal's resolution, the letter of the secretary-general U.N.O. denying the charges of bacteriological warfare in Korea was read. The committee sugested that the Lucknow Medical College be taken over by the Government and the private practice of fully paid staff be stopped.

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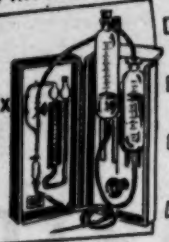

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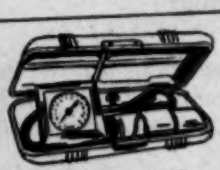

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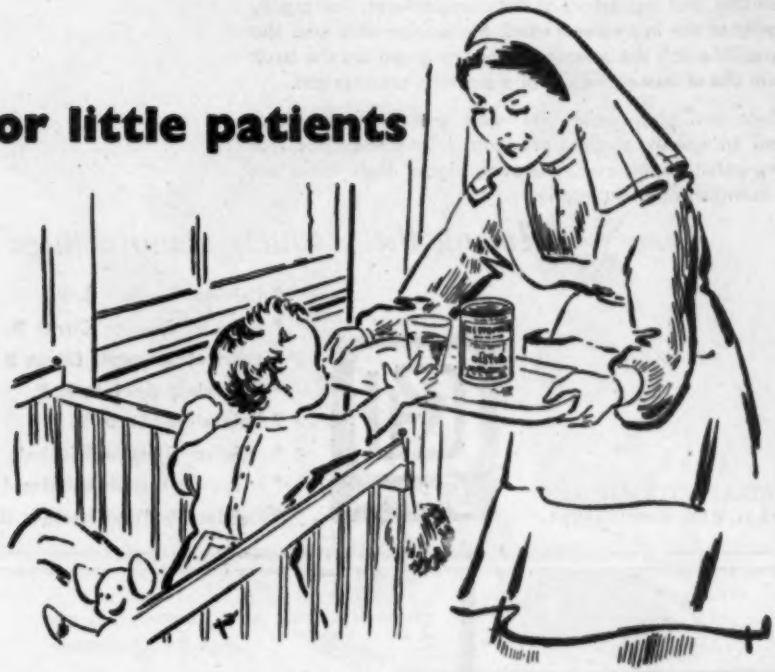

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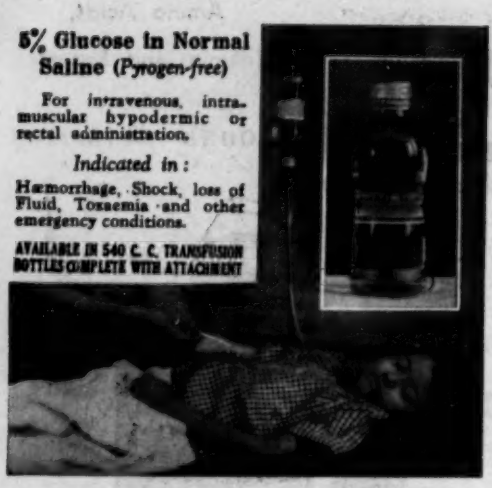

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